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THE ERYTHROCYTE SEDIMENTATION RATE IN PULMONARY TUBERCULOSIS AND OTHER PULMONARY DISEASES.

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THE estimation of the rate at which red blood corpuscles form a sediment in citrated blood, usually known as the "sedimentation test", is a laboratory aid to clinical medicine which is finding increasing application. Though little more than a decade has passed since the test was first introduced, it is now being used in a variety of diseases. Its value has been demonstrated in such diverse conditions as pneumonia, syphilis, cancer, anterior poliomyelitis, pelvic inflammations, acute rheumatism, arthritis, some psychoses, the anæmias, and most important perhaps of all, in pulmonary tuberculosis.

In view of its widespread applicability and its probable acceptance as a routine test in the management of many diseases, it is interesting to review briefly the history of its evolution.

Historical Review.

The sedimentation test is the result of a long series of observations on the blood, dating from the time when Galen, in the second century, first described what is now known as the "buffy coat".[1] Observing the nature of blood from different persons after allowing it to stand in a vessel, he noted that in some a buff-coloured layer of lymph lay above

the sinking red cells. This layer was termed the *crusta sanguinis*. During the next sixteen hundred years the observation of the phenomenon of the buffy coat, as it was called by English scientists, played a most important part in the practice of medicine. The substance of the buffy coat was considered as the *materia morbi*, and on this belief was founded the theory of blood letting.

The reason for the belief is obvious: the buffy coat was found to be constantly present in the blood of persons suffering from illnesses, especially acute diseases. It was also present in the blood of pregnant women. The presence or absence of the *crusta sanguinis* was an important diagnostic feature, even up to the middle of last century. For a detailed and interesting account the reader may refer to the original paper of Fåhraeus.[2]

This Swedish scientist, as a result of his researches, confirmed the work of earlier observers. Hewson,[3] in 1772, had noted that the buffy coat could be formed in two ways, either by delayed coagulation of the blood or by increased velocity in the sinking of the red corpuscles, and he was able to show that the latter was the decisive factor. Later Nasse[4] demonstrated why the velocity was increased; he showed that there is a peculiar tendency in buffy blood for the red cells to cluster. Also he showed that in buffy blood there is an increased fibrinogen content which leads to the formation of rouleaux. It was left to Fåhraeus to discover a final factor in hæmagglutination, namely, an increase of serum globulin.[5] That agglutination depends on the protein content of the plasma is an important fact. Increase of globulin or fibrinogen protein fractions will, therefore, accelerate the sedimentation rate. This increase is found in pregnancy and in most diseases, in the latter as the result of destruction of tissue proteins.

From the foregoing facts Fåhraeus evolved what he has termed the "stabilitary reaction of the blood", or the rate of sedimentation of the red blood cells.

Westergren,[6] an associate of Fåhraeus, in 1921 published an account of the practical value of the reaction. He states that citrated blood placed in a tube and left for one hour reveals a layer of clear plasma above the aggregated red cells measuring about two millimetres in height in healthy males and about five millimetres in healthy females. All workers with his method have since confirmed these as average normal rates, though there is a fairly wide range of normality.

Fåhraeus had noted an increase in the sedimentation rate, that is to say, a reduction in the suspension stability of the blood in pregnancy, infections, malignant disease and in certain psychoses. Westergren decided that it would be "interesting to test the influence of the tuberculosis infection on the stability", and his first papers are based on his application of the test in tuberculosis.

During the past ten years many papers have been written on the value of the test, mainly in tuberculosis, but also in many other diseases.

Objects of Our Investigation.

This paper is the preliminary report of an investigation of the sedimentation rate in pulmonary diseases. Our object was to find, if possible, answers to the following questions: (i) What is the diagnostic value of the test? (ii) To what extent does it aid prognosis and indicate the patient's progress, especially in pulmonary tuberculosis? (iii) Is it of use as a therapeutic guide?

It may be noted here that the apparently discordant results obtained from two patients (Cases I and III) early in our investigation inclined us to attach little value to the test, and for some six months we merely noted readings. A review of our figures after this time led us to an instant appreciation of the value of the test, and it is now established as a routine in the examination of all ward patients under the care of one of us (C.H.) and of all patients receiving artificial pneumothorax therapy at the out-patients' clinic.

Method of Performing the Test.

The majority of workers employ a method wherein blood is obtained from a vein. There are obvious disadvantages to this, and methods have been evolved in which only small amounts of blood obtained from a finger prick are used. Rau[7] and Stolzenbach[8] have shown that results with these methods are just as satisfactory as those obtained with the cumbersome macro-method. The micro-method has also been used by Trail and Stone,[9] Lyle Cummins and Acland,[10] Hermann,[11] Cutler,[12] Linzenmeier,[13] Langer,[14] and others, with slight variations in technique. Most micro-methods require capillary tubes with a graduated length of two hundred millimetres, and a stand is necessary to hold these pipettes. An exception is that described by Lyle Cummins and Acland,[10] in which the tubes and blood volume are small, but in which more apparatus is required; the readings are taken at the end of five hours and twenty-four hours. We have used the method of Langer[14] with the apparatus slightly modified to enable a greater number of estimations to be done at the same time. In Langer's mikro-hæmosedimeter the pipettes are only nine centimetres long, and the whole outfit can be carried in a pocket and used anywhere.

The only solution required is 5% sodium citrate. Small pipettes, 1·5 millimetres in diameter and about nine centimetres long, are marked at B, O and C (see Figure I). The length O to C is graduated in millimetres (0·25). The contents of the pipette are 0·02 cubic centimetre to C, 0·1 cubic centimetre to O, 0·12 cubic centimetre to B. The remaining apparatus consists of an ordinary stabbing needle, a small celluloid plate, and a large porcelain plate, on which are arranged beds of plasticine. The sodium citrate solution is run up into the pipette to the mark C, the patient's finger is cleaned, a small prick is made, and the blood is allowed to run up into the pipette till the citrate solution reaches the top mark, B.

The blood and citrate solution are then blown out on to the celluloid plate and very thoroughly mixed by drawing them up and down the pipette and by stirring the drop with the point of the pipette. This citrated blood is then run up to the mark O on the pipette, and, a finger being held over the top, the tip of the pipette is embedded in the plasticine and it is left standing vertically. The patient's name is marked on the plate and the time is now noted.

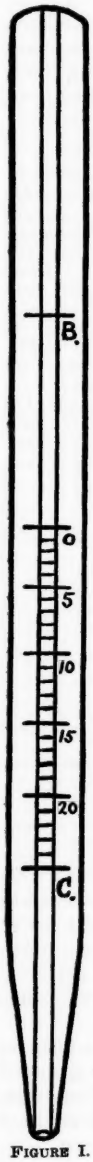


FIGURE I.

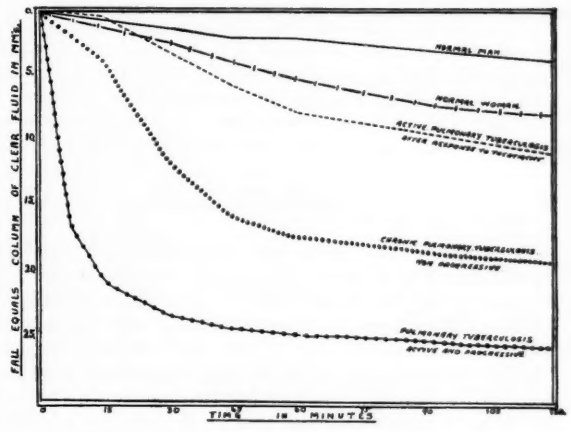
As the red cells aggregate and fall, the plasma is left above, and it is the length of this clear column which is read against the scale at intervals over two hours. The one-hour reading is usually taken to denote the sedimentation rate. It is this figure alone which we have reported in all observations in this paper.

A more accurate measure of the continuous rate of sedimentation of red cells is obtained by taking readings at intervals of fifteen minutes and plotting a graph. This method is illustrated in Graph I.

Before our results are analysed, it should be understood that we have worked under the disadvantage of having a small number of capillary tubes. At first we had only four, though for most of the time we have had nine tubes. More have been ordered from Switzerland to insure their being identical with the original tubes for the purpose of accurate comparison of figures. The literature shows that the length and calibre of the pipettes are far from being standardized, which makes it difficult to compare the results of different workers. For the benefit of those who contemplate employing this test, it may be stated that a tube calibre of 1·5 to 3·0 millimetres will prove satisfactory. A graph has been prepared by Höcker correlating the figures found with two hundred millimetre tubes and the small Langer tubes as used by us.[15]

The limited number of tubes has restricted research in several ways. First, we have tended to select from among tuberculous patients either those undergoing some special treatment or those who are seriously ill. Even so, the numbers in the groups suggest that a fairly reasonable cross-section of pulmonary tuberculosis has been made according to the frequency with which it is met in its varying grades of severity by most physicians. In the second place, it has necessitated a limitation of the study of the sedimentation rate in other pulmonary diseases. These results and the tentative conclusions drawn therefrom can form only a starting point for future investigation. The object of publishing results in an incomplete form is to interest others in a test which has proved a valuable clinical aid.

The sedimentation rate has been determined on 224 patients, with a total of 650 tests.



GRAPH I.
Showing sedimentation rate of erythrocytes over a period of two hours.

Analysis of Results.

In Table I are summarized the results of our observations in 123 patients with pulmonary tuberculosis. Five grades are distinguished and to each grade has been attached a fairly exact prognostic significance. (For brevity, the erythrocyte sedimentation rate will henceforth be referred to as the sedimentation rate.)

Grade I: Sedimentation rate, males to 4, females to 6: No active pulmonary disease.

Grade II: Sedimentation rate, males 4 to 8, females 6 to 8: Slight activity, but tendency to heal; resistance high; prognosis fairly good.

Grade III: Sedimentation rate, both sexes 8 to 13: Activity moderate, requiring careful observation.

Grade IV: Sedimentation rate, both sexes 13 to 18: Considerable activity of disease; tendency to spread. Prognosis poor.

Grade V: Sedimentation rate, both sexes above 18: Resistance minimal, progressive disease, probable early death.

The grading is determined by the initial reading obtained in each case, and the patient's subsequent progress over a period varying from three to fifteen months is noted in the adjacent columns. The period of observation is short, but it gives an indication of the reliability of the test as viewed from the clinical standpoint.

It must be emphasized that the reading may vary greatly in subsequent tests, and the patient may thus rise or fall in grade, with a corresponding change in prognosis. This is the chief value of the test as an indicator of the patient's progress.

Our first table was prepared to determine whether our conclusions from the initial readings were borne out by subsequent clinical events and whether any other information of immediate prognostic value could be deduced from the test.

TABLE I.

Patients with Pulmonary Tuberculosis, graded according to Sedimentation Rate, with their Subsequent Progress.

Grade.	Total Number of Patients.	Condition Quiescent or Arrested.	Condition Much Improved.	Condition Improved.	Condition Stationary.	Condition Worse.	Died.
I	12	6	3	2	0	0	1[1]
II	13	3	3	3	2	1	1[1]
III	31	0	9	7	12[2]	3	0
IV	40	0	8	14	10	5	3
V	27	0	2	2	3	6	14
Totals ..	123	9	25	28	27	15	19

[1]See case notes I and III.

[2]Nine of these have not reported lately; probably a number should be in the "improved" column.

A study of the table will show that the initial reading has a definite prognostic value. Thus in Grade I, six patients (50%) have proceeded to quiescence or arrest of their disease. On the other hand, in Grade V, six (22%) are worse and 14 (52%) have died; none is "quiescent" and only four (15%) have made any improvement. In Grade IV none is yet quiescent and only 22 (55%) have made any improvement. This shows that a patient's chances are better the higher the grade in which he is first placed.

Further analysis of Table I will now be given with illustrative cases.

Grade I: The patient who died was one of those who in the beginning led us to question the value of the test. Brief case notes are as follows:

CASE I.—J.C., male, aged thirty-three years, was admitted to hospital with ascites and enlarged liver. He had advanced bilateral pulmonary tuberculosis, but of no great activity, and he worked up to the time of his admission to hospital. He died unexpectedly three weeks later of sudden myocardial failure. Sedimentation rates at weekly intervals were 3·0, 2·3, 2·3.

Not until we reviewed the literature did we find an explanation for this case. Several workers have noted low figures in the presence of pathological liver conditions. Schulten[16] states that bile acids retard the sedimentation rate, and Westergren,[17] in a long series of cases found the lowest figure (0·8) in acute yellow atrophy of the liver. No other case in this grade calls for comment. A typical one may be quoted:

CASE II (see Graph II).—F.P., male, aged thirty-eight years, was under artificial pneumothorax treatment. Rates have been as follows: May 15, 1931, 3·5; July 15, 1931, 3·0; November 4, 1931, 4·0; May 4, 1932, 4·0. This patient's treatment has been abandoned for six months. The disease is clinically arrested and he is now at work.

In Grade II appears the other case which disconcerted us in our early work.

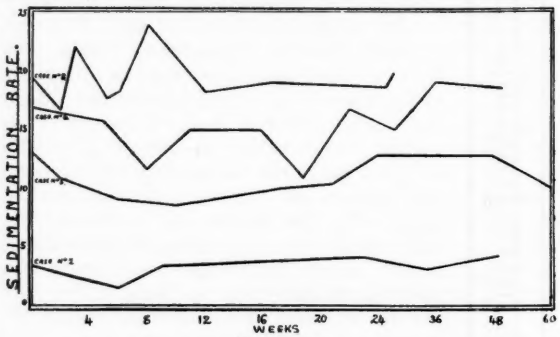
CASE III.—W.H., male, aged twenty-one years, died within thirty hours of admission to hospital with a spontaneous left-sided pneumothorax and extensive right-sided disease. The sedimentation rate obtained was 7·3. He was very cachectic and probably suffering from a considerable degree of alkalosis due to interference with his respiratory exchange.

Several workers have noted that these factors retard the sedimentation rate. This explains why a Grade II figure was found so near death.[11][16]

CASE IV.—One patient, C.J., male, aged thirty-four years, has been in Grade III since seven months ago, the fall synchronizing with a recrudescence of disease in the opposite lung following collapse therapy, when progress was being made towards arrest. His condition, however, has been satisfactory during the last four months. Readings have been: May 18, 1931, 8·0; December 16, 1931, 10·5; May 4, 1932, 8·0; July 20, 1932, 10·0. He is classed as being worse than he was twelve months ago, in view of the X ray findings. Nevertheless he is doing clerical work.

Another patient has now been constantly in Grade I for nine months, and his condition is probably quiescent.

One further patient has fallen to Grade III, but has been observed under collapse treatment for only three months, and her general condition so far is satisfactory.



GRAPH II.

Showing sedimentation rate of patients under observation for periods up to sixty weeks.

Grade III: A guarded prognosis is warranted in the group of 31 in Grade III, only 16 of whom are known to have improved. Two have moved up to the second grade, and their clinical condition accords with this. One patient under artificial pneumothorax treatment, whose disease has become bilateral, has gone down to Grade IV.

Unfortunately, we have not been able to keep in touch with all the patients, and nine are grouped as "stationary" without recent clinical confirmation. Further analysis of this grade will be given in a later paper, when "end results" can be shown.

CASE V.—W.F., male, aged fifty-six years (see Graph II), suffers from diabetes and pulmonary tuberculosis, the latter controlled by artificial pneumothorax. The patient is keeping well clinically and has been at work for some nine months. Readings have been: May 8, 1931, 13·0; July 29, 1931, 8·5; October 14, 1931, 10·5; April 20, 1932, 13·0; May 18, 1932, 10·0.

Diabetes is not a factor in keeping the figure high, so it must be assumed that the pulmonary

disease is still active. Whatever the treatment, the prognosis in a case such as this is doubtful. The sedimentation rate here is a constant reminder, despite the patient's excellent clinical progress (he has gained 12·6 kilograms or two stone in weight), that all is not yet well. We would not at present consider the condition of any patient who remains in this grade as being quiescent, whatever the clinical findings. This statement applies with even greater force to the lower grades. No one in this grade has yet died, nor have any developed obviously progressive disease.

Grade IV is our largest group. Of forty patients only two are able to work. One has fibroid phthisis of long standing, but an anal fistula with recurrent abscess formation is undoubtedly the reason for his presence in this grade. So far, nine patients have risen, five to Grade III and four to Grade II. In none of these does the clinical condition conflict with the sedimentation rate. Four have moved down to Grade V. Three of these are seriously ill. The fourth has just commenced pneumothorax therapy, and his rate has become slower, placing him in Grade IV once more. Many of the remainder have had too few tests to allow us to evaluate the rates as a progress guide. As with Grade III, further analysis will be attempted at a later date. In no case so far have we noted a Grade IV reading when there was definite evidence that the disease was inactive. One patient, who was sent as an "early case" on clinical grounds to a sanatorium, has relapsed and is doing badly. From a study of the cases we are inclined to the opinion that a persistent Grade IV sedimentation rate is a contra-indication to admission to a sanatorium as an "early case". The sedimentation rate may help in deciding the often difficult problem of a patient's eligibility for sanatorium treatment.

Illustrative cases are:

Case VI.—G.H., female, aged thirty years (see Graph II), suffered from unilateral disease treated by artificial pneumothorax; she showed initial improvement, but there was subsequent spread to the other lung, and her condition is now deteriorating. Readings have been: May 20, 1931, 17·0; July 15, 1931, 11·5; September 30, 1931, 11·9; January 20, 1932, 19·0; May 4, 1932, 17·2.

Case VII.—D.C., male, aged thirty-two years, suffers from unilateral disease; he has been under pneumothorax treatment for the last three months. Clinical improvement is evident, with satisfactory collapse of the lung. Readings have been: May 3, 1932, 14·0; May 17, 1932, 15·8; June 8, 1932, 11·6; July 20, 1932, 7·2.

This patient is very anxious to resume work, and it is intended to use the sedimentation rate as the criterion of his ability to work. The value of the sedimentation rate in such cases has been noted by other observers.[18][19] During collapse therapy it is very difficult to decide exactly what is the patient's condition. Physical signs and X ray evidence are lacking; symptoms, with weight and temperature charts, can help us. The sedimentation rate may be added as an additional guide, one of those "ponderable factors which refuse to accommodate themselves to the optimism of the patient or therapeutist".[10] In treatment by artificial pneumothorax we have already found this test to be a valuable guide, on which we place considerable reliance in deciding how much exertion the patient can safely undertake.

Grade V is a melancholy group. Fourteen of the 27 patients have died. Of the remaining 13, four only have risen, two to Grade III and two to Grade IV. Of the former, one has greatly improved as the result of sanatorium treatment and the other has responded well to collapse therapy. The rest all have clinically a very bad prognosis. One case will illustrate.

Case VIII.—E.J., a male, aged twenty-six years (see Graph II), suffered from severe disease, mainly unilateral, with repeated hæmoptyses, following one of which he died. Readings were: May 19, 1931, 22·0; June 11, 1931, 20·0; June 26, 1931, 18·5; July 10, 1931, 24·0; July 24, 1931, 21·5; September 9, 1931, 19·0; November 27, 1931, 20·0.

Our Grade V figures warrant the conclusion that a persistent sedimentation rate above 18 is of very grave prognostic import. A rate of 22 or over would appear to indicate the probability of a fatal ending within a few months. We have not found, as have some observers,[11][16][20] that the sedimentation rate usually falls before death when advanced cachexia is present.

It may be noted that only five patients died while under observation in hospital. The other nine were well enough to leave hospital. Death occurred, however, within a few months.

We conclude from Table I that the sedimentation rate in pulmonary tuberculosis possesses considerable immediate prognostic value. The subsequent analysis demonstrates its usefulness when following the course of the disease in patients under prolonged observation.

We may now turn to Table II. Here is a heterogeneous collection of diseases, mainly pulmonary. We have added figures obtained from "contacts", pregnant women and "normals".

Any detailed discussion on the figures given in this table is not warranted. As in Table I, these are initial figures only. Comment may perhaps be made on some of the groups and on the progress readings of the sedimentation rate in patients under continuous observation. It will be noted that a low or normal figure is the rule in bronchitis. We have not found the rate to be raised to any extent by an acute attack. In bronchiectasis, on the other hand, the rate is usually raised, and great acceleration is seen with an exacerbation. This is consistent with the pathological condition at this time, of a localized pneumonitis surrounding the dilated bronchi. It suggests that a raised sedimentation rate favours the diagnosis of bronchiectasis rather than bronchitis. A persistently high figure indicates involvement of the lung tissue in the suppurative process, and in this way gives prognostic guidance.

In patients with obvious pulmonary suppuration, as would be expected, we have found very high rates. As the acute inflammation subsides, the figure becomes smaller, returning to normal when the lung heals. The progress readings of three

TABLE II.
Sedimentation Rates in Diseases other than Tuberculosis (with the Addition of some Controls).

Disease.	Total Number.	Sedimentation Rate.				Remarks.
		Normal.	Slightly Raised.	Moderately Raised.	Greatly Raised.	
Bronchitis	10	5	4	1		Mostly taken during an acute attack.
Bronchiectasis	11	2	7	2		
Pleurisy with Effusion (Febrile)	4				4	See Graph III.
Pleurisy with Effusion (Afebrile)	4		2	1	1	See Graph III.
Pulmonary Abscess	5			2	3	See Graph III.
Silicosis	6		2	2	2	The four latter have tuberculosis as well.
Neoplasm	2				2	
Suspected Neoplasm	2	1	1			
Hæmoptysis	3	3				1 hysterical; 1 malingerer; 1 under observation, clinically well.
Asthma	2	2				
Chronic Sinusitis	1	1				
Neurasthenia	2	1	1			
Spontaneous Pneumothorax	1	1				Now reexpanded; no evidence of pulmonary disease.
Sepsis	3			2	1	
Diabetes	6	3	3			
Diabetes *plus* Pulmonary Tuberculosis	2		2			
Uræmia	1				1	
Osteoarthritis	2		2			
Pregnancy	6				6	
"Contacts"	7	3	2	2		Mostly young children.
"Normals"	12	10	2			1 suffers from recurrent attacks of malaria (8·5).

patients are shown in Graph III. A return to a normal reading favours a diagnosis of healed lung abscess. Conversely, a persistently raised reading indicates the continuance of the suppurative process. The following cases are typical.

CASE IX: F.B., a female, aged thirty-five years, had an abscess in the right upper lobe, which healed spontaneously. Readings have been: 16·0, 12·0, 12·0, 6·0, 4·5 (see Graph III).

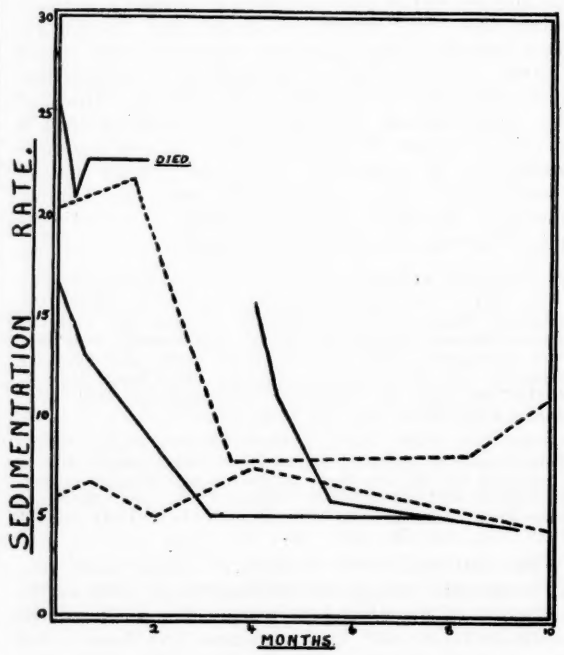
CASE X.—H.S., a male, aged sixty years, has an abscess in the right lower lobe; thoracotomy was performed; the wound breaks down occasionally; there is a large amount of purulent sputum. Readings have been: 15·0, 11·0, 13·5, 19·0, 14·0.

We have noted that a normal sedimentation rate is found in healed pulmonary abscess, even though there is considerable fibrosis, whereas in patients with bronchiectasis, even though they are comparatively well and at work, it is rare to find a normal rate. This is in accord, of course, with clinical findings and illustrates the impersonal prognostic value of the test.

The few readings we have obtained in patients with silicosis suggest that almost normal figures are to be expected in simple silicosis, but that with superadded tuberculosis and the associated tissue destruction, the rate is greatly increased. It is not always easy to determine clinically in silicosis whether tuberculosis is also present; a study of the sedimentation rate may help in the decision.

In pulmonary neoplasm our cases are too few for discussion. In this condition and the pneumonokonioses there is need for further research on the diagnostic value of the test.

The very rapid rates found in patients suffering from serofibrinous pleurisy, especially those with pyrexia, are striking. With defervescence the rate falls, as Graph III illustrates. In some, however, a normal figure was not reached. Our few cases enable us to concur with Roche[20] that "attention to the sedimentation rate during convalescence from pleural effusion should help the physician both to determine the length of convalescence desirable and to regulate the patient's activities".



GRAPH III.

Showing sedimentation rate of patients suffering from pulmonary abscess and of those suffering from pleurisy with effusion. The rate of the former is shown by a continuous line; that of the latter by a broken line.

It has been stated that a more rapid fall is to be observed in children. The few readings we have taken, mostly with "contacts", agree with this finding.

We have endeavoured to observe whether extraneous influences cause variations in the sedimentation rate, thereby lessening its value. It has been stated, for instance, that climate influences the test,[21] and that cold retards the rate.[11] As far as external temperature is concerned, we have not been able to detect any difference in the rate of sedimentation between readings on very cold and hot days, with a temperature range (room temperature) between 31·1° and 12·2° C. (88° and 54° F.), although this is not a very great variation.

Factors such as emotion, hurry, common cold, and a pneumothorax refill, have no effect on the sedimentation rate. Neither does menstruation appear to accelerate the rate, as reported by some observers. It is possible with the method we have employed that minor variations of rate will not be as apparent as when longer tubes are used. This would be a definite advantage in simplifying interpretation. In some of the cases the remarkable constancy of the figures obtained on repeated estimations over a long period has been striking. A case will illustrate.

M.S., a female, aged twenty-seven years, was under pneumothorax treatment. The sedimentation rate at intervals of three weeks was: 9, 11, 9, 10, 9, 8, 8.

It is possible that we may arrive later at a conclusion already held by some observers,[21] that it is not the absolute figure which has the main prognostic value, but rather its progressive alteration in the individual patient. Nevertheless, Table I shows the definite prognostic value of the sedimentation rate on first examination.

The Value of the Test.

Before we summarize our own conclusions, reference may be made to the now extensive literature. The consensus of opinion bears out the work of the original investigators, that the test is a valuable clinical aid in following the course of a disease. Conflict of opinion is revealed in regard to the diagnostic value of the test.

In pulmonary disease most of the work has been done in sanatoria. Here there is general agreement with Roche's summary that "the blood sedimentation rate is of proven value in the management of cases of pulmonary tuberculosis".[20] Clegg[22] and Cutler[23] find the test an accurate measure of the activity of the lesion and as important as temperature, pulse and weight. Trail[24] finds the test of value in determining the duration of a patient's stay in a sanatorium and in deciding on the advisability of continuance of treatment. In this opinion, Wingfield and Goodman[25] concur; they consider, however, that the sedimentation rate has slight value when close clinical observation is available, and suggest that it may be more valuable in clinic and out-patient work.

It is now recognized, of course, that the test is not specific for tuberculosis, and this limits its value in differential diagnosis, though not necessarily its absolute diagnostic value, as we have indicated. Westergren[6] found that the rate was never normal in active tuberculosis, and Clegg[22] noted that every patient with active disease in his series had a raised rate of sedimentation. Cutler[26] found in 5,000 patients with raised rates, only five without any apparent pathological condition. Our results harmonize with these findings. Cutler points out that it may be the last objective observation to return to normal, and that for months it may be the only indication of the pathological state of the diseased lung in artificial pneumothorax therapy.

Weiller[19] regards the sedimentation rate as the alarm signal in artificial pneumothorax therapy, foretelling such complications as the onset of pleural effusion or the spread of the disease in the other lung.

Tegtmeier[18] also finds the test of great value in the control of artificial pneumothorax treatment. He finds, however, no close relationship between the sedimentation rate and the temperature. Linzenmeier,[13] on the other hand, regards it as a more sensitive indication of the activity of the disease than the temperature chart. Léon Bernard[27] considers it to be the best single test yet evolved as an aid to prognosis.

Probably the best paper appearing recently is that by Jacob Cutler, "The Practical Application of the Blood Sedimentation Test in General Medicine". In his summary he writes:

In diagnosis the test has two values, first as a diagnostic lead and the second as a diagnostic gauge. As a diagnostic lead it indicates the presence of serious disease, not infrequently before the disease can be recognized by the usual clinical and laboratory methods. As a gauge of the constitutional disturbance produced by the pathologic process, it indicates the intensity of the disease and thus, like fever or pulse rate or blood count, but more exactly, helps to complete the diagnosis in a qualitative sense.

As a prognostic index and, similarly, as a guide in treatment, the rate of sedimentation has been shown to be a more accurate and reliable reflection of the real condition of the patient than our usually accepted procedures. It invariably becomes more rapid as the disease progresses, and approaches stability only as the physical condition returns to normal. It is not influenced by psychologic factors and may be the only warning of disaster among promising clinical signs, such as gain in weight and the absence or subsidence of symptoms. In this way it is often the only, or the most accurate, evidence of favourable, or of unfavourable, response to treatment.[26]

Summary.

The rate of sedimentation of the red blood cells has been determined on 224 patients suffering from pulmonary tuberculosis and other diseases. An analysis of the figures has been made in order to assess the diagnostic and prognostic value of the test.

Conclusions.

1. The erythrocyte sedimentation test performed with a minute volume of blood, and employing the one-hour reading, is entirely reliable and is very easily carried out.

2. The sedimentation rate has a limited but definite diagnostic value, in indicating the presence or absence of serious inflammatory disease.

3. The sedimentation rate in pulmonary tuberculosis (taken in conjunction with the clinical

symptoms) gives a reliable indication of the activity of the disease.

4. The sedimentation rate has a definite prognostic value in observing the course of chronic disease associated with tissue destruction.

5. The sedimentation rate is of definite value in observing the result of treatment, especially in artificial pneumothorax therapy.

6. Further work is required in order to determine how far the test is of assistance in the differential diagnosis of pulmonary diseases.

Acknowledgements.

Our thanks are due to Dr. A. J. Hood Stobo, for his skilful drawing of the pipette and the graphs; and also to Dr. W. Wilson Ingram and Dr. V. M. Coppleson, for their helpful criticism.

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THE MEDIAN PRESSURE AND HYPERTENSION MOYENNE SOLITAIRE SYNDROME: SOME NEW ASPECTS OF OSCILLOMETRY.[1]

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TWENTY odd years have passed since the introduction of Pachon's oscillometer into the realms of clinical medicine. During this time it has found favour with French physicians, who have done a considerable amount of research work, the outcome of which has been the discovery of the median pressure. This, we feel, marks as big an advance in the study of hypertension and cardiac function as the first introduction of instruments for recording the blood pressure.

At the present time there are four methods of ascertaining the blood pressure: (i) Auscultatory. This is supposed to give fairly accurate readings for both systolic and diastolic pressures. (ii) Palpatory. This method is less reliable than the auscultatory and does not give any correct diastolic figures. (iii) Oscillometric. This is the method under discussion. (iv) Plethysmometric. This is obtained by means of the plethysmosphygmo-oscillometer of Spengler and Guillaume. There are two cuffs, one is fixed round the arm and the other, which is attached to the oscillometer needle, is placed round the forearm. As the blood reaches the arteries of the forearm it causes an expansion of the limb, which in its turn gives rise to an alteration in pressure in the cuff, causing a deflection of the needle. Readings are taken as with the oscillometer.

The Oscillometer.

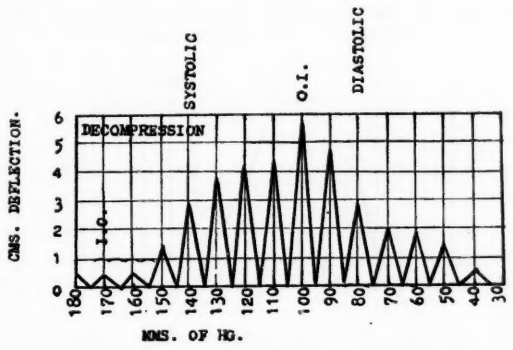
The oscillometer is very sensitive, and furthermore, its sensitivity is remarkably constant. On account of extreme sensitivity, the oscillometer has become of great use, not only in merely obtaining the systolic and diastolic pressures, but in the study of the function of the cardio-vascular system. The importance of its use by the surgeon and obstetrician, as well as by the physician, will be discussed later.

The instrument is constructed as follows. An aneroid capsule is enclosed within a rigid and perfectly air-tight metal box. The box, the capsule and the cuff are normally all in communication. A

[1] Read at a meeting of the Queensland Branch of the British Medical Association on August 5, 1932.

pump is attached to the box for the purpose of raising the pressure within the circuit. This is registered on a manometer attached to the box, and a compression in button form is provided for disconnecting the aneroid capsule from the main box. When this is done, the pulsations from the vessels are transferred by means of the cuff along the connecting tubing, through the aneroid, and thence to the recording needle.

The pressure within the system is raised to a point above the expected systolic pressure of the patient. When the button is pressed there are only small deflections of the recording needle. These small deflections are equal in height and are caused by the wave in the artery impinging against the occluded portion of the vessel. These are known as initial oscillations, usually designated I.O. on the graphs (see Graph I). As the pressure is lowered



Graph I.

to a point where the blood is just able to traverse the previously occluded portion of the vessel, there is a sudden increase in needle deflection. This point is the systolic pressure. As the pressure is further lowered, the deflections increase to a maximum and then decrease. The first steep descent represents the diastolic pressure.

The figures are plotted with abscissæ representing millimetres of mercury and the ordinates representing centimetres of deflection. The oscillometric curve is formed by connecting these points.

The greatest deflection of the needle was termed by Pachon "the oscillometric index". This is produced when the counter-pressure of the armlet equals the pressure within the artery. At this moment the arterial wall is in a fashion floating, and the change in form which it undergoes on the passage of the pulse wave is exactly proportionate to the amplitude of this wave, which, in its turn, indicates the force of the heart beat. This figure, according to Pachon, is of much greater importance than the simple reading of systolic pressure. Billard, however, made no attempt to replace the systolic pressure readings with that of the oscillometric index, but he insisted that much can be gained from the study of both figures. We will discuss the minimum pressure later in the light of recent research.

The index, according to Pachon,[1] is a measure of two conditions: (i) arterial and (ii) cardiac.

All things being equal in the case of the artery under examination, the index represents the strength of the cardiac impulse. At this time the wave traversing the artery encounters the least degree of resistance from the vascular system.

All things being equal, when the cardiac impulse is being measured, the index indicates the state of relaxation or spasm of the vessels under examination.

When feeling a pulse, it is not always possible to judge the force of the heart beat by the strength of the pulse wave. A wave of certain force in a vessel in a state of spasm gives only a small pulsation, whereas the same wave in a relaxed vessel gives a far greater pulsation. Consequently, a vessel in a state of relaxation would tend to give a full pulse, even with a poorly acting heart. Therefore, the height of the oscillometric index is no real guide to the cardiac function.

The oscillometric index is in reality the total of all the partial expansions of the various arteries which give rise to it. The index is thus a direct indication of the peripheral circulation, both from the point of view of the vascular tone or vasomotor activity and the arterial permeability. Therefore, if we know that either the arterial or the cardiac factor is constant, a variation in the index must be due to a variation in the other factor. On account of these two factors it is not possible to gain any information of value by merely comparing the index of two different persons.

At the same time the character of the pulse can be noted, especially with regard to alterations in force and rhythm. *Pulsus alternans*, frequently not noted on palpation, can be detected by means of the oscillometer, and a distinction can often be made between doubtful cases of auricular fibrillation and extrasystoles.

Systolic Pressure.

We sometimes find that the systolic pressure recorded by the oscillometer is twenty to thirty millimetres higher than the figure obtained by the sphygmomanometer.

Astier[2] quotes the work of Fabre and Delmas-Marsalet, who demonstrated in the following manner the reason for these findings. They transilluminated the nail beds so as to watch the reappearance of the capillary circulation after complete disappearance due to compression of the brachial artery by an arm band. They state that in a normal subject, aged twenty-three years, the capillary circulation reappeared at a pressure of 150 millimetres of mercury, whereas the systolic pressure obtained by the Riva-Rocci instrument was only 120 millimetres of mercury. In another normal subject, twenty-one years of age, they noted three readings as follows: (i) Stoppage of the capillary circulation, 140 millimetres of mercury; (ii) maximum pressure obtained by the oscillometer, 140 millimetres of mercury; (iii) maximum

pressure with the Riva-Rocci instrument, 110 millimetres of mercury.

These experiments prove that the higher readings of the oscillometer represent the correct maximum pressure required to occlude the vessel completely, whereas by the sphygmometric methods the figure is under-estimated.

Meye and Laubry, after similar experiments, concluded that the oscillometric maximum corresponded to the reappearance of a sluggish circulation in the capillaries without dilatation, and that the sphygmometric maximum corresponded to pressure which allowed a filling of the capillaries. This is supposed to have been overcome by the use of a double cuff. However, we still find that a certain number of patients give oscillometric readings approximately 10% higher than the sphygmometric figures.

The Problem of the Minimum or Diastolic Pressure.

Gomez,[3] in a masterly article, pointed out that researches aroused by the study of the median pressure have led to a revision of sphygmometric ideas, particularly with regard to the minimum pressure. Does this really correspond to the moment when, in the course of a progressive decompression, the oscillations give way to oscillations of less size, coinciding with the period at which in auscultation or palpation there is a peculiar vibration in the vessels below the arm band?

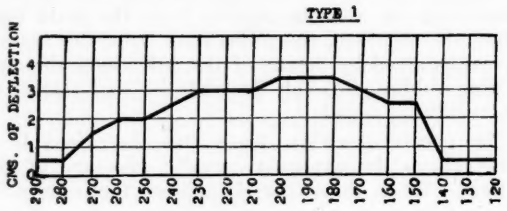
The minimum pressure is the expression of the limit below which, in a given segment of the arterial tree, the pressure can no longer fall, that is, its minimum.

MacWilliam and Melville,[4] in England, first noticed in a scheme of the circulation that the minimum pressure was contemporaneous with the moment at which, under an increasing counter-pressure, the oscillations, at first weak, suddenly increased in size. Pachon later stated that this pressure coincided with the commencement in the course of a progressive decompression of a terminal and distinct zone of oscillation with a characteristic slope.

Gomez has, in animal experiments with Gley, confirmed this fact. The counter-pressure for which one observes the appearance of a terminal zone of oscillations with characteristic slopes, is contemporaneous with the minimum pressure. Gomez collected three hundred curves on subjects from Vaquez's clinic, some normal and others with various cardio-vascular lesions. In studying these as a whole, and especially the sequence from the largest oscillation down to zero, one sees at once that there is a great variation in type. Our own observations confirm this point.

The types of variation can be classified thus: (i) A typical form in which it is possible to fix definitely the minimum pressure (see Graph II). (ii) A tracing which is much less definite and measurement is more or less guesswork (see Graph III). (iii) There is no alteration of the tracing and measurement is impossible (see Graph IV).

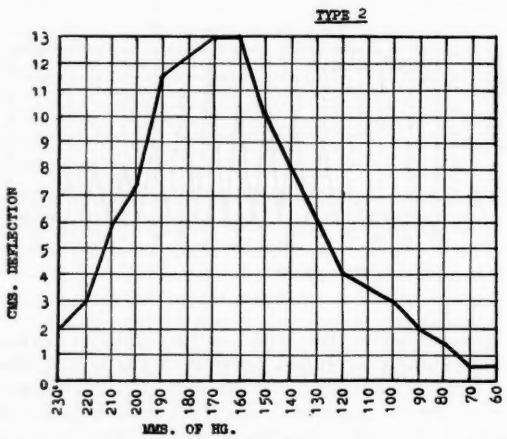
The reading was easy in 27·3% of the three hundred curves, and in the remaining 72·7% it was a mere guess, or impossible.



GRAPH II.

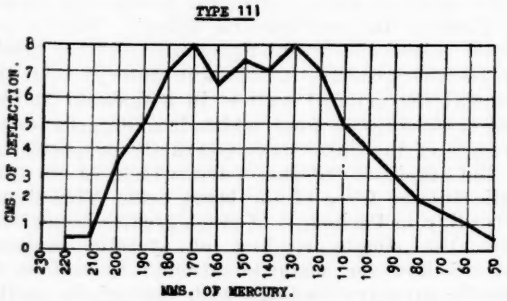
In our series of two hundred cases, 30% fell into the first category and 70% into the second.

Thus, as Gomez states, we may conclude that the reading of the diastolic pressure seems to be a snare and delusion. The next question is, do we



GRAPH III.

get the desired result by the palpatory and auscultatory methods? Take successful oscillometric tracings which by numerous animal experiments have been undoubtedly proved to be correct, and compare them with the auscultatory and palpatory



GRAPH IV.

figures; they never correspond. Sometimes they approximate to one another, especially in aortic insufficiency, but more often they are intermediary

between the median and minimum pressures without corresponding to either. This was borne out in comparing numbers of our cases.

Fabre, of Lille, after a careful study of the sphygmometric bruits, has also arrived at the same conclusion. Thus, Gomez asserts that if the oscillometer is able to show us the minimum pressure in a restricted number of cases, the auscultatory and palpatory methods are quite unable to do so.

From these observations we learn that the minimum pressure in the healthy adult is from 50 to 70 millimetres of mercury, most usually 60. It is generally higher in the upper limbs than in the lower, and higher in the forearm than in the arm, and in the ankle than in the thigh.

Pachon also demonstrated that the diastolic pressure varies according as the armlet of the sphygmomanometer is placed at a level above or below that of the heart. When one diminishes the local pressure ten millimetres by elevating the arm, the diastolic pressure falls approximately one millimetre; conversely, increasing the local pressure increases the figure by approximately the same amount.

The figure is raised with age, varies with menstruation, and undergoes variation during different periods of the day. At from twenty to fifty years of age the figure is 50 to 70 millimetres of mercury; from fifty years onwards it is 60 to 80 millimetres.

Technical difficulties have made the study of the effect of physical exercise impossible. Our own observations confirm this finding, as the curves often change from a type from which it is easy to read the minimum pressure to one from which the pressure cannot be read.

Pachon has recently repeated a statement to the effect that the minimum pressure was remarkably constant throughout the whole of the arterial tree. He here referred to the contrapressure corresponding to the greatest oscillations which we now know to be, not the minimum, but the median pressure.

Median Pressure.

Vaquez,[5] Gley and Gomez sum up the known facts concerning the value of the systolic and diastolic readings obtained at first by the Riva-Rocci instrument and later by the oscillometer. They also point out that Cawadias[6] has demonstrated the further importance of the oscillometer in the diagnosis and prognosis of arterial diseases. To these the authors add a further and important use of the oscillometer, namely, the determination of the median pressure. Previously, the only means of determining this pressure had been the introduction into an artery of a compensatory manometer of Marey. This, of course, is not practicable in man.

The median pressure is not the mean pressure between the two extremes of pressure, namely, the systolic and diastolic levels. This is purely an arithmetical mean and has no clinical importance. The median pressure is the constant pressure which would assure the same delivery of blood in the vessel as the variable pressure which prevails there. As an analogy, the median of an alternating current would be a constant current which would discharge the same energy as the variable current. Pachon, in order to obviate confusion, has termed this the median dynamic pressure.

In 1921, Pachon, experimenting with a special circulatory scheme to which he had joined a Marey compensatory manometer, discovered that the oscillometric index corresponded to the median pressure. This was only obtained schematically, and there was no proof that it actually occurred in the living individual. In 1931, Gley and Gomez[7] performed experiments on the living animal, the results of which agreed entirely with Pachon's findings and proved that the oscillometric index and the median pressure accurately corresponded.

Before describing the experiments, we shall give a description of Marey's compensatory manometer. This is a single mercury manometer, with a narrowing at the lower portion of the recording arm. When the ordinary manometer is connected to an artery, the pulsations are transmitted through the bowl to the column of mercury. In this type, however, the constriction at the base damps down any oscillations and the column of mercury rises and stays at a fixed level, which is the median pressure.

In the experiment, the oscillometer and the compensatory manometer are connected to the same vessel and an oscillometric curve is taken. In this way it is found that the oscillometric index and the median pressure correspond. The figure obtained for dogs, about 110 millimetres of mercury, is much higher than that for human beings. In man the median pressure is usually in the vicinity of 90 millimetres of mercury, a figure which was previously regarded as the diastolic level. The experiments of Gley and Gomez on animals demonstrated that the minimal or diastolic pressure was much lower and is in the region of 50 to 60 millimetres of mercury. Almost twenty years ago Potain, by an elaborate calculation, obtained a figure for the median and diastolic pressures, almost the same as that obtained in the way just described. No confirmatory findings were produced until the work of Gley and Gomez in 1931.

In the study of arterial pressure a search has frequently been made for a constant figure which would be a gauge of the functional capacity of the heart. The variations of the systolic and diastolic figures exclude both these readings from being suitable for the purpose. However, it is reasonable to suppose that a normally functioning heart must have some constant pressure. This stable figure exists in the median dynamic pressure, which represents the factor hitherto sought. Vaquez, Kisthinios and Papaioannou[8] have recently performed experiments in a large number of cases which prove this fact.

Age.—Median pressure in normal individuals increases slightly with age, as shown in the accompanying table.

Age in Years.	Millimetres of mercury.
10-25	80- 90, most usually 90
25-50	80-110, most usually 90
50	90-120, most usually 100

Thus from youth to old age the average median pressure does not vary more than 10 to 15 millimetres of mercury, which contrasts with the much greater variability of the systolic and diastolic figures.

Sex.—Unlike the systolic pressure, which is lower in females than in males, the median pressure is the same in both sexes.

Digestion.—During the process of digestion there is a varying rise and fall of pressure, both systolic and diastolic, but the median pressure remains absolutely constant.

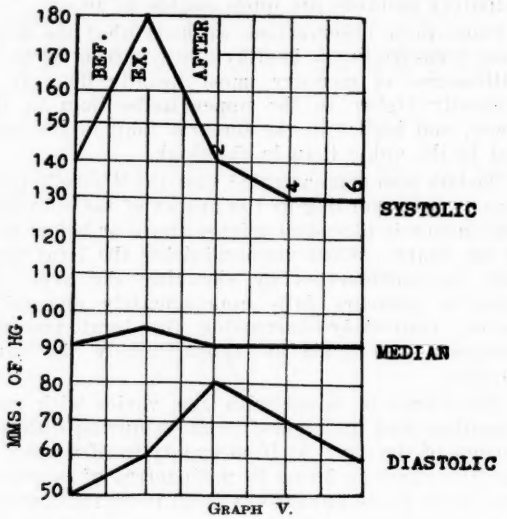
Exercise.—The effect of exercise in causing an elevation of systolic pressure and a less pronounced rise in diastolic figures is well known. Observations carried out by Gomez and Lajoie at the same time as Vaquez, Kisthinios and Papaioannou did their work, demonstrated conclusively that the median pressure during exercise does not vary more than five millimetres of mercury and usually not at all. Thus, in a healthy subject, moderate exercise does not change the vascular regimen as represented by the median pressure.

Other conditions, such as plunging the hands into hot and cold water, which cause definite alterations in the systolic pressure, have no effect on the median pressure in a normal person.

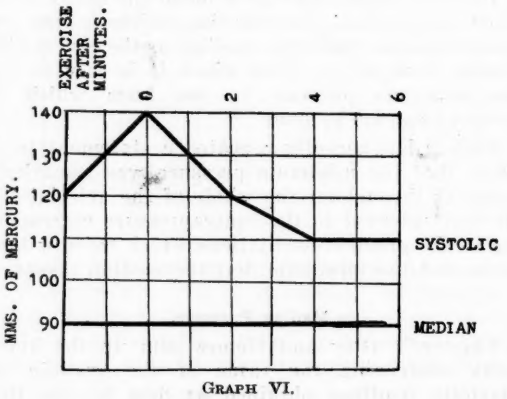
In cases of aortic insufficiency there is a high pulse pressure, and the pressure in the upper limbs is lower than that in the lower limbs, but the median pressures remain in the vicinity of 90 millimetres in both upper and lower limbs. This explains why so many patients with aortic insufficiency can carry on with a very large valvular leak. In spite of the great variation in systolic and diastolic figures, the median alone of all the various pressures which represent the functional activity of the heart, remains at normal level in both upper and lower limbs.

From the time that it was possible to measure the human pressure it was thought that a means had been obtained for measuring the functional capacity of the heart. The systolic pressure was first made use of, but it was found that many individuals with raised blood pressure had normally functioning hearts, whilst a normal blood pressure was frequently present with a failing heart. Similarly, the diastolic pressure was estimated, but proved to be an unreliable guide.

Gomez and Lajoie[9] studied a number of persons with normal tension and hypertension, in order to determine how the median pressure reacted to exercise. Their conclusions with regard to normal subjects have been mentioned previously, to the effect that severe exercise, for example, running two hundred metres, has no effect, or at the most, causes an elevation of only five millimetres of mercury, with an immediate return to normal. In hypertension with no evidence of cardiac failure, the effect on the median pressure following moderate exercise is the same as in normal individuals. In all cases both the systolic and diastolic figures show a definite rise (see Graphs V, VI).



GRAPH V.



GRAPH VI.

However, patients manifesting clinical evidence of the decompensation have a definite rise in the median pressure. In decompensation the median pressure approaches more closely to the systolic level and not the diastolic level, as in subjects with well compensated hearts. Failing hearts manifest a rise in median pressure following exercise. Gomez and Lajoie studied twenty-two patients showing clinical evidence of cardiac insufficiency, and divided them into two groups. The first ten patients showed no elevation of the median pressure after moderate exercise. These all suffered from right-sided failure without any left ventricular involvement. There was a failure of the pulmonary circulation only, as evidenced by dyspnœa, cyanosis, hepatic congestion, œdema of the legs, and dilata-

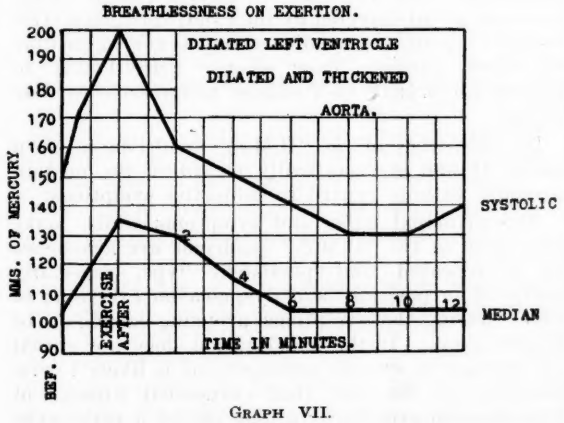
tion of the right side of the heart; thus there was no reason why the normal pressure variations should have been altered.

The remaining patients showing a definite rise of 20 to 30 millimetres of mercury in the median pressure following exercise were all suffering from left-sided failure, as evidenced clinically and radiologically. Thus the authors conclude that with the normal heart there is no rise in the median pressure after exercise, or if there is a slight rise of five millimetres, the return to normal is immediate. When there is a definite rise or a slow return after exercise, there is a state of decompensation of the left ventricle.

Objection may be raised that this elevation is due to an acceleration of the pulse. How can this be so when a failing right side of the heart gives just as pronounced an increase in pulse rate, but no rise in the median pressure? The important point is not only the degree of elevation of the pressure, but, as with all diseased organs, the slow return to normal which is often present, for example, in diabetic curves.

It is too early to state that an increase in median pressure following exercise is the first warning of a failing left ventricle, but it is a sign which must not be neglected.

We are carrying out experiments which show that even violent exercise in a patient with a healthy heart causes only a minimal rise in the median pressure. In a failing heart the median pressure rises to a greater height and falls more slowly to normal (Graph VII).



Graph VII.

In some oscillometric curves a distinct plateau is present in which it is impossible to detect any highest point. In the early stages of research it had been customary to take as the median pressure the centre of the plateau. Vaquez, Gomez and Lajoie[10] demonstrated that by using a large cuff approximately fifteen centimetres wide, the plateau curve was obviated. They also showed that the centre of the plateau was not the median pressure, but was as much as 10 to 20 millimetres above or below the correct reading. This has been obviated in the instrument recently produced by Vaquez's clinic, which we shall describe later.

A female, aged thirty-nine years, two years previously had an induction of labour at eight months on account of albuminuria and hypertension. Five weeks ago there was a miscarriage at ten weeks, preceded by a rise in blood pressure. The question was whether she should become pregnant again.

At the first examination the systolic pressure was 186 millimetres of mercury and the diastolic 114. The median pressure was 125 millimetres of mercury. The next day the readings were systolic 180 millimetres, and diastolic 104, with the median still 125. Two days later the systolic was 150 and the diastolic 96 millimetres of mercury, the median still remaining at 125 millimetres of mercury.

	Systolic.	Median.	Diastolic.
May 30, 1932 ..	184	125	114
June 1, 1932 ..	180	125	104
June 3, 1932 ..	150	125	96

The median pressure remained fixed at a definitely raised level in spite of the apparent normal readings for systolic and diastolic figures.

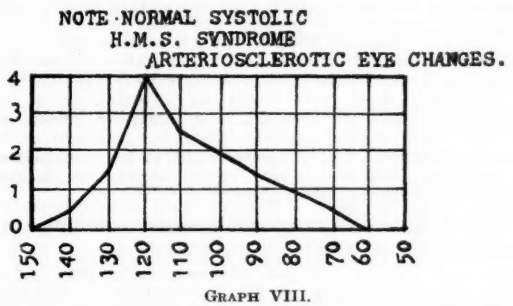
The kidney function was slightly impaired, as evidenced by blood urea, urea concentration *et cetera*.

Hypertension Moyenne Solitaire: "H.M.S." Syndrome.

Vaquez and Gomez[11] describe the *hypertension moyenne solitaire* syndrome, which must take its place beside hypertension, whether paroxysmal or permanent, in the classification of hypertensive disease. Its main sign, the elevation of the median pressure, can alone reveal unsuspected morbid states or explain their beginning and their cause.

It is difficult to find a suitable translation for the word *"solitaire"*, so for convenience we termed this the "H.M.S." syndrome, of which we have seen several examples.

Case I.—A patient reported by the ophthalmic surgeon to have all the eye signs of hypertension showed no elevation of the systolic pressure. This patient had a median pressure of 120 millimetres of mercury. Sphygmometric readings did not reveal any elevation of either systolic or diastolic readings (see Graph VIII).

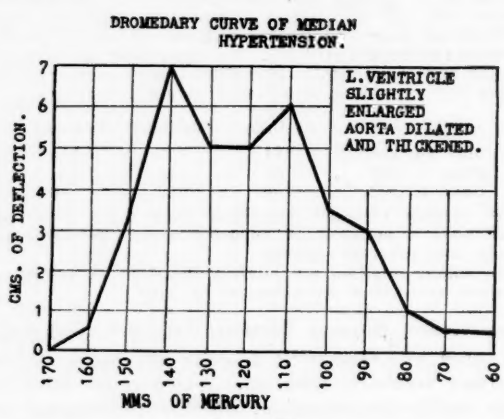


Graph VIII.

These patients have been explained as having had a raised pressure which, for some unknown reason, has fallen to normal. This hardly seems a rational explanation, especially as no cause for the fall has been given and the raised median pressure, sustained over a period, more readily explains this condition.

Case II.—A male patient, aged fifty-six years, complained of headaches, breathlessness and of becoming easily fatigued. There was pallor and a slight degree of anæmia. The urinary system was clear. The sphygmometric readings for the systolic pressure was 150 millimetres of

mercury and for the diastolic 85 millimetres of mercury. The figures were well within normal limits for the first examination. The median pressure, however, was 140 millimetres—a definitely raised figure. The aortic second sound was accentuated. An orthodiagram revealed a hypertrophied left ventricle and dilated and thickened aorta. There was a sustained increase in the median pressure after exercise, to 150 millimetres of mercury, indicating some failure of compensation of the left ventricle (see graph IX).



GRAPH IX.

CASE III.—A female patient, aged thirty-nine years, complained of headache for four years, becoming much worse and occurring almost every day. The headaches were usually in the left parietal region and were often present in the morning. There was no history nor other evidence of sinus disease. The eyes had been tested and glasses prescribed without any improvement. The urine was clear and renal function unaffected. The hæmoglobin value was 90%, with a normal blood smear. The radial vessels were thickened and palpable. The systolic blood pressure was 160 millimetres of mercury and the diastolic pressure 100 millimetres. On percussion there was dulness behind and to the right of the upper part of the sternum. The left border could not be determined accurately, on account of large breasts. An orthodiagram revealed increase in diameter of the aortic arch and descending aorta, with generalized increase in density, also hypertrophy of the left ventricle, with an increase of 2·2 centimetres in the transverse diameter of the heart. In other words, the changes of hypertensive cardiac disease were present to a far greater degree than would be expected from the moderate increase in systolic and diastolic pressures. However, the median pressure was 140 millimetres of mercury, compared with a normal of 90 to 100 millimetres, which is quite sufficient to give rise to the cardiac changes and headaches which were present.

Vaquez and Gomez describe some examples as follows.

The first patient referred to them on account of a raised systolic pressure of 230 millimetres of mercury, was found on examination to have a pressure of 155, not greatly increased for a man of thirty-seven years of age. Radioscopically, the heart was definitely hypertrophied, the hypertrophy involving particularly the left ventricle. The median pressure, however, was found to be 130 millimetres, a figure considerably above normal, thus confirming the original diagnosis of a definite hypertension.

The second case is as follows.

A young woman of twenty-eight years complained of violent headaches, yielding only momentarily to antipyrin, of defective vision, terminating finally in a transitory amaurosis. One would naturally think of a hypertensive syndrome in this case. However, the sphygmometric readings were 145 millimetres of mercury systolic and 85 millimetres diastolic. The only abnormal sign consisted in an augmentation of the second sound at the base, which was difficult to ascribe either to the aortic or pulmonary areas.

Dr. Bailliart reported that the fundi showed a typical hypertensive retinitis. This was difficult to explain with a systolic pressure of only 150 millimetres of mercury. However, the median pressure was 140 millimetres of mercury, a very considerable increase. This patient had a hypertension, but a median hypertension alone.

The orthodiagram showed marked enlargement of the left ventricle and increase in size and density of the aorta.

CASE IV.—A woman, aged thirty-six, complained of severe and constant occipital headaches, of mists in front of the eyes, of dizziness and buzzing in the ears, all symptoms which are common in hypertension. The systolic pressure was 150 millimetres of mercury, but the median pressure was 130, a figure considerably above normal.

The interesting point about the case was the fact that several months later the systolic pressure rose to 190 millimetres and became fixed at that level without any alteration in the symptoms.

These cases demonstrate clearly the extreme importance of the median pressure, not only to the physiologist, but also to the clinician. Previously we have shown that both in the pathological and physiological states the median pressure has a characteristic fixity. Life is compatible with large variation of the extreme pressure as long as the median pressure remains more or less constant. On the contrary, changes in the latter, even though the extreme pressures remain normal, bring in their wake profound disturbances of the circulatory *régime*.

This new type of hypertension is not rare and may be discovered by chance in apparently benign conditions, or just as likely in a syndrome arousing suspicions of the existence of a hypertension which, however, is not detected by the usual sphygmometric means. Again, the condition may occur in cardiac disorders, ranging from simple arrhythmias to *angina pectoris* or to a sudden progressive cardiac failure.

The discovery of the "H.M.S." syndrome is often made, if one systematically measures the median pressure without regard to subjective symptoms.

The principal signs and symptoms which make one think of the "H.M.S." syndrome are headaches of a repeated and persistent type, especially occipital in position, and dyspnœa on exertion; in other words, the symptoms arousing suspicion of hypertension. If the examination does not reveal an increase in systolic pressure, one is likely to err, forgetful of the fact that evanescent attacks of hypertension precede by a long period a permanent increase in pressure. This is the time at which it is important to study the median pressure, as it is the only diagnostic feature at this stage.

Unlike the paroxysmal attacks of systolic hypertension, the median pressure, when once increased, remains fixed, and in the increase may be very considerable, without a parallel rise in the systolic pressure. The next, but relatively unimportant, point is to try to cause a reappearance of the systolic hypertension by exercise. Normally, there is an increase in the systolic pressure up to 60 milli-

metres of mercury. These patients, however, show up to twice the normal increase, causing a reappearance of the systolic hypertension which was absent at the time of examination.

On auscultation in median hypertension there is a ringing aortic second sound, and radiologically an increase in size and density of the aorta, together with left ventricular enlargement, which, at this stage, is only slight.

Finally, a most curious fact is the occurrence of a camel-back type of oscillometric tracing. This type, without doubt, is due to some abnormal effort of the heart during the whole evacuation period, extending from the time when the pressure is at its lowest to a time when it is at its highest point, a period corresponding especially to the median pressure. This type of curve occurs in *hypertension moyenne solitaire,* but the hypertension can exist without the characteristic curve.

We have observed this type of curve in two other patients with hypertension, both with a systolic pressure well above 200 millimetres of mercury. These two cases support the view that the curve is caused by some abnormality of the heart, probably a poorly acting myocardium.

The first patient, with a median pressure between 130 and 170 millimetres of mercury, had previously had several attacks of cerebral thrombosis, and his general circulatory condition was very poor.

The second patient, with a median pressure of 155 millimetres of mercury, seen one week later, gave sphygmometric readings of 165 millimetres of mercury, systolic pressure, and 135 millimetres, diastolic; the pulse pressure was only 30 millimetres of mercury, indicating poor heart force. Unfortunately, the median pressure was not taken on the second occasion.

These facts, particularly the occurrence of raised median pressure without any symptoms, demonstrate the importance of the median pressure readings in examining subjects for insurance. Up till now, readings of the extreme pressures have been sufficient; but the fallacy of merely relying on these figures has, we feel, been amply demonstrated.

The study of the median pressure is tending to give the true significance to various cardiac lesions previously regarded as innocent, and to increase the significance of hypertension in its relationship to them. A certain percentage of individuals presenting extrasystoles and attacks of paroxysmal tachycardia are regarded as having a harmless disorder, especially in the absence of any increased pressure or electrocardiographic evidence of muscular disability.

The first patient quoted by Vaquez and his collaborators was a man, aged fifty-six years, who had had extrasystoles for thirty years without any detectable cause. Recently, attacks of paroxysmal tachycardia had supervened. The electrocardiogram showed evidence of grave myocardial weakness at this stage. At no time had there been an elevation of the systolic pressure, but at the time of examination the median pressure was 140 millimetres of mercury. There was a radioscopic evidence of considerable cardiac enlargement, with increase in size and density of the aorta.

It is surely more correct to ascribe these findings to an increased median pressure sustained over a long period than to a myocarditis or primary hypertrophy of the heart.

A second patient with paroxysmal tachycardia had, in the apparent absence of a detectable abnormality in the cardio-vascular system, been treated as a patient with functional disorder of the heart. The systolic pressure was 145 millimetres, but the median was 125, a figure definitely raised in spite of the normal maximum reading.

Several other similar cases are quoted by the authors illustrating a similar condition. The serious end-result observed in Case I predicts a bad prognosis for the other patients seen earlier in the evolution of the disease.

Several authors have ascribed the cardiac hypertrophy in these cases to the irregularities present because there has been no other obvious cause. These observations must be regarded as throwing an entirely different light on the order of events. A more feasible explanation is that the raised median pressure leads to cardiac hypertrophy, which is the cause and not the result of the irregularities.

Potain and Starling have both shown that difficulty in emptying is definitely a cause of cardiac hypertrophy. This disability may be situated in the heart itself, as in aortic valvular lesions or in an increased peripheral resistance. Everyone has seen cases of hypertrophy in which none of the mentioned causes were detectable. In these the condition has been termed essential cardiac hypertrophy, primary cardiac insufficiency and myocarditis. Surely it is more logical to suppose that there is a similar unsuspected cause, that is to say, an increased peripheral resistance. This unknown factor has now been proved to be a raised median pressure, which is a sure sign of an increased peripheral resistance.

The first case illustrating this point, described by Vaquez and Gomez, is that of a young man who was thought to have chronic bronchitis and emphysema. There was a history of cough, but no pyrexia. The first onset was with effort dyspnœa. There were a few râles in the chest, but not sufficient to account for all the symptoms. X ray examination revealed an enlargement of the left ventricle and not of the right, as would be found in cardiac enlargement secondary to lung obstruction. The extreme pressures were normal, but the median was 125 millimetres of mercury. This is sufficient to explain the cardiac hypertrophy, which, in the absence of the knowledge, would have been described as essential.

Let us recall the case described previously in which the electrocardiogram showed grave myocardial damage. There again there was no systolic hypertension to account for the hypertrophy, but the median pressure was 140 millimetres of mercury.

Cordier, Veil and Collomb have reported similar findings in Lyons. The discovery of the "H.M.S." syndrome throws a new light on the origin, ætiology and development of arterial hypertension. Until now we have recognized hypertension only as an established disease without knowing when or how it commenced. Noting the long duration of its

development, we have supposed that it must commence early and sometimes be hereditary and familial.

The recognition of the "H.M.S." syndrome allows an early diagnosis to be made during the silent period and long before it is manifested by indisputable signs or symptoms. Several cases have been reported in patients under thirty years of age. One patient, a man, aged twenty-eight years, with glaucoma, had a median pressure of 130 millimetres of mercury and a markedly enlarged heart and aorta. One cannot doubt that such abnormalities in these individuals are the beginning of an arterial hypertension which will not become fully apparent for a very long time.

The familial and hereditary influence, suspected for a long time as a basis of hypertension, is shown up in its true light by an analysis of several cases of "H.M.S." syndrome.

One patient had a brother who suffered from the same symptoms and who had died of cardiac insufficiency; his mother had died a hemiplegic at the age of fifty-three years.

Another patient had one brother with a similar condition, a sister with angina, and a young brother who had all the symptoms of hypertension, occipital headache, visual disturbance and giddiness *et cetera*, but whose pressure had not been ascertained.

If one had measured the systolic pressure alone in these cases, the patients would have been considered so far free from the familial taint, but they were in reality already definitely affected.

Many observers, noting the familial tendency towards death in the fourth or fifth decade from such catastrophes as cerebral hæmorrhage, sudden heart failure and cardio-renal sclerosis, have concluded that if doubt exists as to the presence of a cardiac heredity, then a vascular heredity is indisputable. Previously, this tendency has only revealed itself slowly after an apparently healthy life, but this hereditary taint is already present in a latent state in the first years of life. The case of a young man, aged seventeen years, was quoted.

This lad, whose mother was suffering from a median hypertension, with all its signs and symptoms, had a normal systolic pressure, but a median pressure of 130 millimetres of mercury. X ray examination revealed no increase in cardiac diameter, but an aorta with the opacity of old age. His grandmother had a very enlarged heart, with complete irregularity of a mitral insufficiency and a dilated aorta, signs easily attributable to an arterial hypertension, although the arrhythmia interfered with the correct pressure readings.

The previous description now permits us to trace the evolution of median hypertension. Quite often, very early, even during adolescence, the raised median pressure is present. This is followed by a long, semi-silent period, during which cardiac arrhythmias, occipital headaches or glaucoma may develop. At this stage there is usually a definite enlargement of the left ventricle and an increase in size and opacity of the aorta, frequently termed juvenile or essential. These conditions continue for some years till the occurrence of sudden catastrophes, such as cerebral hæmorrhages, cardiac rupture or failure, which are common in arterial hypertension.

On the other hand, when the systolic pressure becomes raised, the condition takes on the true picture of hypertension, which appears to have commenced at that stage, but which, in reality, is only the end-result of a long existent morbid state. This change has been noted in one case previously recorded, and doubtless, as more patients are followed, the number will be very considerably increased.

In all cases of "H.M.S." syndrome no fault was found in the kidney function, an important observation against the renal origin of hypertension.

The Median Pressure in Eclampsia and Glaucoma.

The discovery of a raised median pressure in eclampsia and glaucoma without a parallel rise in the systolic pressure has helped to place them more definitely in the field of hypertensive disorders.

In the course of their investigation, Levy, Solal, Kisthinios and Lapage noted that in more than 80% of normal cases the median pressure did not undergo any change during pregnancy and the puerperium. There is usually a slight rise up to ten millimetres of mercury during labour, but this is evanescent and the normal level is reached within an hour of delivery.

Following this, they carried their investigation further to study the changes in the median pressure during eclampsia. Since the first observations of Vaquez and Nobecourt in 1897, it is generally accepted that eclampsia is accompanied by elevation of the arterial pressure. This fact has been confirmed by numerous authors since that date. Vaquez has insisted that if the hypertension is already evident in the days preceding the eclampsia, it usually reaches its highest degree at the time of the onset of the crisis. It is possible almost with certainty to predict the next appearance of the fits and it is not possible to be certain of their complete disappearance until after the return of the pressure to a normal level.

However, several authors have drawn attention to the fact that hypertension may be absent in eclampsia. This has been confirmed by Levy, Solal, Kisthinios and Lapage, during the course of their observations on the median dynamic pressure. In the opinion of Vaquez this absence of hypertension has not been real, but due more to the lack of adequate means for detecting its presence. This has been amply borne out by the above authors, who quote some interesting and instructive cases.

CASE I.—A *primipara*, aged twenty-one years, had a normal delivery followed four hours later by an eclamptic fit, followed closely by a second. Routine examination near full term revealed an absence of albumin, but a systolic pressure of 178 millimetres of mercury and a median pressure of 150 millimetres. After the second fit the systolic pressure fell to 146 and the median to 120. Within five days both the systolic and the median pressures had returned to normal.

CASE II.—A *primipara*, aged twenty-one years, passed urine which contained much albumin at the eighth month. The patient also complained of severe headaches and of a mist in front of the eyes. The systolic pressure was slightly increased to 158 millimetres of mercury, but the relative increase in median pressure was much greater,

namely, from a normal 90 to 130 millimetres of mercury. Half an hour after delivery an eclamptic fit occurred. Two hours later the systolic and median pressures were respectively 136 and 90 millimetres of mercury. Some hours later there was a recurrence of headache, when the median pressure was increased to 110 millimetres of mercury, but the systolic pressure remained unchanged. For the next two weeks the median pressure remained at 110, when a thrombosis developed in one leg. After this cleared up, during the ensuing three weeks, the median pressure returned to 95 millimetres of mercury.

From their observations, it is to be noted that before and during eclamptic seizures the median pressure undergoes considerable elevation in spite of only moderate elevation of the systolic level.

Couvelaire quotes two interesting cases with regard to the prognostic value of the median pressure in eclampsia.

The first patient was admitted to hospital in coma and had four fits after admission. The systolic and diastolic figures were not raised at any time; on the contrary, the median pressure was raised to 120 millimetres of mercury on admission and showed a steady and continuous rise to 150 millimetres of mercury shortly before death.

The other patient had definitely increased systolic and diastolic figures. These remained practically unchanged, whilst the median pressure showed a gradual decrease after each fit, from 160 to 110 millimetres of mercury.

Two facts emerge from the study of these cases: firstly, that the median pressure does not run parallel to the systolic and diastolic figures and secondly, the prognostic value of the median pressure. In the first case, which was fatal, there was a steady and continuous rise to 150 millimetres of mercury, whereas the second patient, who recovered, had a gradual fall in median pressure.

In the new-born, when it is not possible to detect heart sounds, circulation in the extremities may at times be detected with the oscillometer.

Just as interesting are the studies of Baillard and Gomez[12] on the relation between glaucoma and general circulatory disturbances. For many years it had been the opinion of Vaquez and Gomez that arterial hypertension played a leading rôle in the ætiology of glaucoma. However, although hypertension had been noted in a number of cases, the systolic pressure in others was not raised above the normal level.

Baillard and Gomez have studied the median pressure in glaucomatous patients with normal systolic levels. In seventeen cases the systolic pressure was abnormally raised in ten, the median pressure running parallel with the systolic. In six others the systolic pressure was normal, with a median pressure ranging from 120 to 140 millimetres of mercury. In one case in particular, that of a young man, aged twenty-eight years, with a normal systolic pressure, but a median pressure of 130 millimetres of mercury, the heart and aorta were both definitely enlarged. Rollet, investigating the question, came to the same conclusions.

One patient, whose condition had at first been diagnosed as glaucoma, was found to have a normal median pressure. On further examination the primary diagnosis was proved to be erroneous.

We have had the opportunity of observing only three cases of glaucoma. In two patients, aged fifty-five and sixty years, there was a definite hypertension with median pressures of 145 and 130 millimetres of mercury respectively. In the third patient, aged thirty years, there was no increase in systolic or diastolic figures, and the median pressure was normal.

Use of the Oscillometer in Surgery.

In 1927, Samuels,[13] in America, called attention to the extensive use of the oscillometer in France. By this means it is possible to detect the degree and level of vessel obliteration in the limbs.

When it is necessary to ligate the main vessel, it is important to know if a collateral circulation is present to carry on the nutrition of the limb. This can be determined by taking oscillometric readings before and after the compression of the vessel at the proposed level of ligation.

Silbert and Samuels,[14] also in America, in an article on *thromboangiitis obliterans*, discussed the prognostic value of the oscillometer. At the level at which the cuff is applied, the oscillometer measures the expansion of the limb due to pulsation of the vessels. The use of the oscillometer often reveals pulsations when none can be felt on palpation.

One hundred and twenty-four patients were followed for a period of from three months to four and a half years. These had been divided into two main groups: Group I, in which the oscillometric reading at the ankle was 0·5 or more, and Group II, in which the reading was 0 or less than 0·5. The prognosis in Group I was good. There had not been any amputations, although gangrene was present in some. In Group II the outlook became progressively poorer as the clinical condition was more advanced. All the amputations occurred in the latter group.

Comment.

We wish to add a few words concerning the oscillometer. When the readings are being taken, the needle does not return to zero after each deflection, so that a varying starting and end point have to be observed. This is very difficult, especially with a rapidly beating heart and large oscillations. The introduction of the kymometer of Vaquez, Gley and Gomez has obviated this difficulty by the introduction of a valve which allows the needle to start from zero each time. This instrument also obviates the plateau curve already mentioned.

The most important points we wish to stress are:

1. The recording of the median pressure is such a useful guide to cardiac function.

2. The presence of a new syndrome, isolated median hypertension, which opens up a new sphere in the search for a cause of arterial hypertension and which reveals the ætiology of cardiac hypertrophy without any discoverable cause.

Conclusions.

1. The oscillometer is of extreme value in the study of hypertension and allied disorders, and seems destined to replace all sphygmometric appliances.

2. The systolic and diastolic pressures are too variable and inaccurate to be of much real value in the study of hypertension, especially in regard to its early stages.

3. The median pressure owing to its constancy and precision, is of paramount importance in studying circulatory phenomena.

4. Oscillometry throws new light on the early stages of left ventricular failure.

5. The syndrome of "H.M.S." (*hypertension moyenne solitaire*) is a discovery of great importance in vascular disease and appears to be destined to be of considerable practical utility in obstetrics and surgery.

6. Routine oscillometry is important for diagnosis and prognosis in vascular diseases of extremities.

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Reports of Cases.

AN UNUSUAL INTESTINAL CONDITION.

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ON January 31, 1932, at 6 a.m., Mrs. A.Y., a *primipara*, gave birth to a male child weighing 3·1 kilograms (seven pounds). The birth was normal and unaided, the presentation was by the vertex. The child appeared normal and passed urine immediately. It slept quietly all day and passed urine frequently, but no meconium.

Beyond a little crying at intervals in the early morning of February 1, the child appeared to be normal. About 8 a.m. it began to cry more freely, with some apparent pain, and the nurse noticed the abdomen becoming swollen. An enema was given, but could not be retained. The return contained some thick whitish mucoid material, but no sign of meconium, blood or flatus.

When seen about 9 a.m., the child was in obvious pain, with a distended abdomen, and across the upper part of the abdomen, from the right costal margin to the left costal margin and down into the left lumbar region, was seen what was thought to be the distended large bowel, but no peristalsis was visible. During the examination the child vomited a watery, greenish fluid for the first time.

Dr. W. W. Feather, of Roma, was called in consultation, and the condition was considered to be intestinal obstruction, due probably to intussusception, but, during the interval between my examination and his arrival, a matter of two hours approximately, the child's condition had become so serious, with superadded congestion of the lung bases, probably resulting from inspiration of the vomitus, that it was considered too late for any surgical interference, even under local anæsthesia. The child gradually sank and died at 3.45 p.m.

Permission was obtained to open the abdomen after death, and a very rare condition was found. All abdominal organs were normal except the intestinal canal.

From the pylorus to approximately the middle of the small intestine the gut was acutely congested and gradually became distended, as traced distally, where it ended blindly, the blind end being dilated to the extent of 3·1 centimetres (one and a quarter inches) in diameter.

At the pylorus the wall of the gut was red and gradually became darker in colour as the increased distension occurred, till at the blind end it was greenish-black and contained a lot of dark green fluid.

Then, continuing on in the free side of the mesentery was another half of small intestine, of a size of about a diameter of six millimetres (one quarter of an inch), continuous distally with caecum, appendix and large bowel.

The large intestine was collapsed and resembled a cord of a slightly larger diameter than the small intestine. The proximal free end of this latter half of small intestine was lying attached to the distended blind end of the proximal half of the small intestine, in the free edge of the small mesentery, at a distance of 1·25 centimetres (half an inch) and resembled a small normal adult appendix.

This is an interesting condition when considered with the normal development of the intestinal canal from a single duct.

CARCINOMA OF THE SIGMOID AND ENDOMETRIOMA IN THE PELVIC POUCH AND VAGINAL FORNIX.

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AND

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THE following case of cancer of the sigmoid, together with endometrioma of a sole remaining ovary and vaginal wall in the posterior fornix, is of interest owing to the clinical resemblance of endometriomatous deposits to secondary carcinomatous deposits in the pelvis, which would have precluded radical excision of the carcinoma of the sigmoid.

In January, 1931, J.D., a married woman, aged forty-one years, was sent for opinion and treatment, with the following history.

She had suffered from chronic constipation for many years. Eight years previously she had been told that the constipation was due to retroversion of the uterus, and she had had an operation for replacement, and a right oophorectomy was done for a cystic ovary. This relieved the constipation for five years. During this period she had a healthy child, and three years later a miscarriage at the third month of pregnancy. This was followed by an attack of glomerular nephritis, for which she was under treatment for eighteen months. During this period the constipation returned, progressed and finally became accompanied by tenesmus, a feeling of pressure in the rectum, loss of weight (a stone in three months) and anæmia. She did not seek medical advice for this until in December, 1930, her symptoms culminated in an attack

of acute intestinal obstruction. At a country hospital a left iliac colostomy had been done. A distended coil of sigmoid was opened, as the lower end of a lump had been felt *per rectum* and had been looked on as a rectal carcinoma.

When she had recovered from this, she first came under our observation for further treatment, with a well functioning colostomy.

Under anæsthesia she was examined and sigmoidoscopy was performed. A typical ring carcinoma was seen in the distal limb of the sigmoid, fifteen centimetres (six inches) from the colostomy opening and a little above the pelvi-rectal junction. A piece was removed by a punch forceps for microscopical section, and the diagnosis was confirmed. The rectum was normal, but a hard lump, well defined and 2·5 centimetres (an inch) in diameter, was felt anterior to the rectal ampulla. Vaginal examination showed this to be in and under the tissue of the vaginal wall in the mid-line of the vaginal fornix posteriorly. Also, a mass the size of a golf ball was felt at the site of the left ovary. This was not fixed, nor was it attached to the sigmoid carcinoma. As the mass in the vaginal vault, was unusually small and too limited to be a carcinomatous deposit in the pouch of Douglas invading the vaginal vault, an alternative of a coincident endometrioma was considered. Examination of a small piece removed from this *per vaginam* for microscopical section revealed endometrioma.

As there was no other evidence of metastases, laparotomy was done a few days later. There were no deposits in liver, pelvis or peritoneum, and the carcinoma of the sigmoid was resected, together with several enlarged paracolic glands. It was impossible to reestablish the colon after adequate removal, so the cut ends were infolded and the colostomy was left to function. The left ovary was enlarged and contained many endometriomatous cysts (this view was confirmed later by microscopical section) and was excised. The right ovary was absent.

When the wound had healed it was considered that deep X ray therapy would be beneficial, first of all because the endometrioma had not all been removed, and secondly because it would be a prophylactic to the surrounding lymphatic area beyond that removed. The treatment was given in split doses, and commenced on March 5, 1931, with the following factors: A "C.P." deep therapy machine was used, with 200 kilovolts, four milliampères of current, a filter of 0·75 millimetre of copper and 2·0 millimetres of aluminium, and a distance of 50 centimetres, with large fields. A total of 650 *r* units was given on each of four fields to the pelvis and lower part of the abdomen. Some diarrhœa and vomiting resulted, with œdema of the colostomy wound.

On September 14, 1931, a further course was commenced. This was identical with the previous course except that 850 *r* units were given on each field. Some redness of the skin appeared in about eight days, followed by an even bronzing.

From this time she steadily regained weight and strength and on examination three months later there was no sign of the mass in the vaginal wall, and now (June, 1932) she is in normal health and able to do her housework.

Reviews.

THE NEW "BRITISH PHARMACOPŒIA".

On September 30, 1932, by notice in the *London Gazette*, the "British Pharmacopœia, 1932,"[1] became official and was published in place of the 1914 issue. In Australia it will become authoritative after the necessary statutory gazette notices. The price of the new issue is twice that of the previous edition, and it contains 130 more pages. The intervals between successive pharmacopœias in the past have been irregular, varying from three to eighteen years. It is recommended that in future revision of the pharmacopœia should be made every ten years. The present issue is the sixth since the *Medical Act* of 1858. In the compilation of the pharmacopœia assistance was rendered by various subcommittees of pharmacy and of pharmaceutical chemistry, and by pharmacological and clinical experts. Close contact has been maintained with the American authorities in their revision of the "United States Pharmacopœia".

The hypodermic injections of the 1914 pharmacopœia have been omitted, it being considered that the potent or dangerous substances contained in them should be prescribed in quantities by weight and not in terms of volume of a solution. The wines are also omitted in conformity with the article of the International Agreement which requires that "no potent drug shall be prepared in the form of a medicinal wine". Hence the old established ipecacuanha wine disappears, to be replaced by a tincture. Such infusions as have been retained have been renamed fresh infusions. Some concentrated infusions have been introduced; but sanction has not been given for the use of a concentrated infusion of digitalis. Aromatic waters are renamed distilled waters. The strong and weak tinctures of iodine have been renamed strong and weak solutions of iodine, in conformity with the article of the International Agreement requiring that "simple solutions of chemical substances shall not be named tinctures". In regard to suppositories, a general method of preparation is described, the practitioner being left to state the quantity of the drug which he wishes incorporated in each. At the same time, standard suppositories are retained for use when the amount of the drug is not indicated. An entirely new feature is the introduction of *oculenta* or eye ointments, containing atropine, hyoscine, physostigmine, cocaine, yellow oxide of mercury and iodoform. Industrial methylated spirit is described and its use, under definite conditions, allowed in the preparation of some specified liquids for external use and in the preparation of certain medicines for internal administration, if the material be so treated as to eliminate methylated spirit from the final product.

Since the 1914 pharmacopœia was issued some therapeutic agents have become established in medical practice which can be assayed only by biological methods (antitoxins, sera, tuberculin, organic arsenical compounds, insulin and extract of the posterior lobe of the pituitary body). Provision is made for the control of these drugs by the *Therapeutic Substances Act*, 1925. Some such preparations are in the new pharmacopœia. The new pharmacopœia also describes biological methods of assay for such drugs as digitalis and strophanthus (and their preparations) and strophanthin. The standard material adopted in the new pharmacopœia for tincture of strophanthus is not ouabain, but is a standard tincture of strophanthus, the activity of which has been determined in relation to that of the international standard, ouabain. Biological tests for vitamins *A* and *D* in cod liver oil are not included. These vitamins vary greatly in different samples, all of which may be of great use therapeutically. A biological assay for squill is not included. Aconite is not deemed of sufficient medicinal value to warrant the inclusion of a biological method of assay.

It has not been deemed advisable as a general principle to adopt the Continental practice of weighing both liquids and solids. The provisions of the International Agreement of 1930 have been studied and, where practicable, the requirements of the new pharmacopœia have been made to correspond with them. When any divergence occurs, the points of dissimilarity are indicated. The monographs give the Latin title (such as *Anethum*), the official abbreviation (*Aneth.*), the English name (Dill), synonyms (*Anethi fructus*, dill fruit), chemical formulæ and molecular weight (when known), the source, characters of the preparation, tests for identity and purity, assay and doses. The official abbreviations seem rather superfluous, but are obviously included to prevent confusion when different drugs have somewhat similar titles. The letters I.A. after a synonym indicate that the synonym is the title employed in the International Agreement. Tests for identity do not, in every case, suffice to establish proof of identity, and tests for purity are not framed to

[1] "The British Pharmacopœia, 1932. Published Under the Direction of the General Council of Medical Education and Registration of the United Kingdom." London: Constable and Company, Limited. Medium 8vo., pp. 763. Price: 21s. net.

provide against all possible impurities. The doses mentioned are not authoritatively enjoined as binding on practitioners, who must exercise their own judgement and act on their own responsibility in regard to the amount of any therapeutic agent prescribed or administered. In the case of potent drugs, such as strychnine, after the description of the preparation there is indicated the amount of the active agent in a given dose. Thus solution of strychnine hydrochloride contains in 0·8 mil 0·008 gramme, and in twelve minims about one-ninth of a grain of strychnine hydrochloride, 0·8 mil (twelve minims) being the maximum dose. Doses are expressed both in the metric and imperial systems, the equivalence being only approximate. In prescribing it is recommended that symbols be discarded. The drachm symbol is often used to represent sixty grains or one-eighth of an ounce, which is quite different. It is also used to indicate one fluid drachm. The ounce symbol may represent 480 grains or 437·5 grains or a fluid ounce. It is recommended that the prescriber cease to employ these symbols, and when the imperial system is used, solids should be prescribed in grains and ounces, one ounce being 437·5 grains. Liquids should be prescribed in minims and fluid ounces. The quantities should be written in Arabic, not Roman numerals. The letter G. should be used for grammes, and gr. for grains. Prescribing in the metric system is impracticable at present in Australia. Technically it would be illegal, as standard weights and measures are not officially recognized. Any prescription in the metric system would have to be converted into the imperial system and dispensed in officially stamped weights and measures. The inaccuracy of the "Table of Equivalents" is clearly pointed out. Thus a gramme really is a little over 15·4 grains, and a mil or cubic centimetre is nearly 16·9 minims. In the table of equivalents one gramme or mil is taken as equal to 15 grains or minims. A milligramme may be considered equal to either one-sixty-fourth or one-sixtieth of a grain. The equivalents are only for the convenience of the prescriber and are not accurate enough for pharmaceutical or other purposes. Quantities which can be expressed in convenient figures in one system will not yield figures equally convenient in the other system, if expressed in exact equivalents. A useful innovation in the monographs is the list of preparations containing the drug inserted at the end of the monograph, for example, *nux vomica*, camphor, and so on. Substances used only as reagents in testing are described in an appendix and not included in the principal list.

Omissions.

Drugs and their preparations to the number of 357 have been omitted. The most noteworthy are: acetanilide, dilute hydriodic acid, aconitine, ammonium bromide, benzamine lactate, subnitrate of bismuth, butyl chloral hydrate, citrate of caffeine (replaced by the sodio-benzoate), *cannabis indica*, tartarated iron, gelsemium, hyoscyamine, lithium salts, phosphorus, salol, scoparium, strontium bromide, tincture of aconite, and the acetate, carbonate and valerianate of zinc. Some of the liquors are omitted on the ground that the potent or dangerous substances contained in them should be prescribed in quantities by weight, not in terms of volume of a solution. Many substances of merely local usage have been omitted. These may be included in addenda for the countries concerned, to be drawn up by the local authorities. Thus many vegetable products indigenous to Australia and elsewhere have rightly been excluded as being solely of local interest. *Kino eucalypti* ("red gum") is an example. Our Australian medicinal leech also disappears, as well as *spermaceti*, derived from the sperm whale, which inhabits Australian waters. Unfortunately, the omissions do not go nearly far enough. The new pharmacopœia is still loaded with superfluous simple and aromatic bitters, vegetable astringents and vegetable purgatives. In other directions also more ruthless elimination is still desirable. With the additional quinine salts there are too many quinine and cinchona preparations. It is understood that the discarded preparations will be incorporated in the next British Pharmaceutical Codex (B.P.C.). If so, that unfortunate volume will become hopelessly unwieldly. The last issue (1923) contains 1,669 pages.

Additions.

The additions, including the *oculenta*, number 134. The most important are acriflavine, amidopyrine, ethylene, sodium antimonyltartrate, amylocaine hydrochloride ("Stovaine"), soluble barbitone, barium sulphate (for diagnosis), carbon dioxide, carbon tetrachloride, carbromal ("Adalin" or "Uradal"), chlorbutol, cinchophen ("Atophan"), emetine hydrochloride, emetine and bismuth iodide, ephedrine hydrochloride, ergotoxine ethanesulphonate, injections of precipitated bismuth, salicylate of bismuth, iron, mercury and calomel, insulin, neoarsphenamine (but not arsphenamine or the silver compound), oil of chenopodium, hydnocarpus oil, phenobarbitone (and soluble phenobarbitone), sulphate of quinidine, strophanthin and sulpharsphenamine. Pituitary (posterior lobe) extract is introduced with a dosage in units, not in volume; solution of irradiated ergosterol is also included, with prophylactic and curative doses in units and minims. The antitoxins are those for diphtheria, tetanus and gas gangrene. There is also antidysentery serum, vaccine lymph, antityphoid-parathyphoid vaccine, old tuberculin, Schick control, Schick test toxin and diphtheria prophylactic. Totaquine is a mixture of alkaloids from the bark of a certain species of cinchona.

The names of many drugs and preparations have been altered. Compound tincture of camphor (paregoric) becomes camphorated tincture of opium. Trinitrin (nitroglycerin) becomes glyceryl trinitrate, and arsenious acid becomes arsenic trioxide. The composition of many preparations has been altered; *liquor arsenicalis* (Fowler's solution) is now neutral. In some instances the strength of the preparation has been changed. Fresh infusion of digitalis is intended to contain, in 120 mils (four fluid ounces), six units of activity. The single dose of all digitalis preparations has been conspicuously increased.

The appendices deal, *inter alia*, with determination of pH values, quantitative tests for lead and arsenic, determination of iodine value of fats and fixed oils, antimony trichloride test for cod liver oil, biological assays, and special processes used in alkaloidal assays.

The indigenous flora of Australia is represented in the new pharmacopœia by the tamarind, which occurs on the north-west coast; cajuput oil, the product of the broad-leaved tea tree or paper bark tree; and eucalyptus oil (species not now indicated). In both cajuput and eucalyptus oils the cineole requirements have been increased in conformity with American standards. Eucalyptol is also included, to contain not less than 97·5% of cineole. A further addition is oil of Australian sandal wood (*Eucarya spicata*), indigenous to Western Australia, with not less than 90% of free alcohols.

Notes on Books, Current Journals and New Appliances.

THE UNIVERSITY OF LEYDEN.

DR. R. W. INNES SMITH has written an unusual book entitled: "English-Speaking Students of Medicine at the University of Leyden."[1] Dr. J. D. Comrie, Lecturer on the History of Medicine at the University of Edinburgh, has written a foreword in which he points out that during the last half of the seventeenth and the earlier half of the eighteenth century Leyden was the chief resort of Englishmen, Irishmen and Scotsmen who wished to attain eminence in the profession of medicine. Dr. Innes Smith has taken as the basis of his investigations the album of students at Leyden University from 1575 to 1875. He gives an alphabetical list of the students, with their qualifications and a short account of their achievements. This book will be of interest to medical practitioners devoted to the history of medicine.

[1] "English-Speaking Students of Medicine at the University of Leyden", by R. W. Innes Smith, M.D., with foreword by J. D. Comrie, M.D., F.R.C.P.; 1932. Edinburgh: Oliver and Boyd. Royal 8vo., pp. 280. Price: 16s. net.

The Medical Journal of Australia

SATURDAY, OCTOBER 1, 1932.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

THE RIGHTS OF THE PATIENT.

Not many weeks ago an article was published in these columns dealing with the general practitioner and the consultant who is a member of the honorary staff of a public hospital; particular reference was made to patients who are sent to a hospital by a general practitioner in order that they may receive special treatment. It was shown that members of hospital staffs have advantages of many kinds, not possessed by other practitioners; the suggestion was made that the rights of the general practitioner are sometimes ignored by members of hospital staffs, and attention was drawn to the need for recognizing these rights in the present state of hospital practice. This is an important subject and one which should be understood by both general practitioner and consultant. If medical practice is to be peaceable, harmonious and progressive, it must be conducted on a basis of mutual understanding by all types of practitioner. If it is to be successful in the true sense of the word, it must also be tinged with altruism, flavoured with a love of humanity and leavened by a desire for service. In other words, the patient has certain rights which have to be considered. Only one of these will be discussed at present.

When a patient comes to a medical practitioner for treatment, he comes because he thinks that the medical practitioner will be able to make a correct diagnosis and will offer advice likely to result in disappearance of symptoms. Though some are anxious to give their illness a name, most patients are chiefly concerned with symptoms. According to the measure of relief they obtain, so do most patients cease worrying about their ailments and rest content with the advice their medical attendants have given them. A large percentage of people who are attacked by illness must be regarded as abnormal from the mental point of view—they become introspective and sometimes their whole mental horizon is taken up by their aches, their pains and their distressful feelings. Even the most level-headed people in the community may have their whole mental outlook changed by disease. The mental effect of bodily disease is not recognized as it should be recognized, and even when it is known to exist, is often ignored by the medical attendant. Human nature being what it is, and the effects of disease being so diverse, it is not surprising that patients frequently want a second or even a third opinion in regard to either diagnosis or treatment. The patient has a perfect right to obtain as many opinions as he likes regarding his condition. This right is not ceded by all medical practitioners. It is quite a common thing for patients to be censured in no uncertain way by their regular medical attendant when, acting as most people unaccustomed to medical usages would act, they seek an opinion from a consultant and either tell the medical attendant what the consultant has said or bring a letter from him setting out his views and offering certain advice.

Unfortunately every medical practitioner is not a student of human nature. The wise man will forestall his patient and will suggest that another opinion be sought. Patients should be taught that the opinion of a consultant will be of much more value to them if they take a letter to the consultant from the regular attendant, that the consultant will send a report to the medical attendant, and that the treatment decided on can be carried out under his supervision. They must be shown that the

report will be much more intelligible to the medical attendant than to them, that the medical attendant will keep the report, and that it will be available and most likely useful in any later developments of the illness or in any subsequent emergency. It will, however, be useless trying to make patients adopt the correct procedure if medical practitioners show resentment when a second opinion is sought. The medical practitioner exists for the patient, not the patient for the medical practitioner.

Current Comment.

ANTHELMINTICS.

Some hundreds of drugs have been employed as anthelmintics. This of itself is sufficient to warrant a healthy scepticism concerning the merits of any one of them. The fact that new anthelmintics are constantly being discovered is further evidence that none of the old ones is entirely satisfactory. There is no drug that can be guaranteed to cure a patient completely of infestation with any particular species of worm; and there is no efficient anthelmintic in common use that is free of toxicity. Some of the drugs in common use are dangerous even to life; carbon tetrachloride, used freely in the treatment of ankylostomiasis, has caused death to certain persons possessing an idiosyncrasy to it; the administration of oil of chenopodium, the drug most commonly used in the treatment of ascariasis, has also proved fatal. Certainly, as far as is known, the mortality rate is very low; but it is an unhappy thought that death can occur at all as the result of such a simple procedure as the administration of a vermifuge. It is possible that the mortality rate is much higher than the apparent rate revealed by available statistics; much of the treatment is the so-called "mass" treatment of many thousands of natives, who, perhaps, are not seen by a medical practitioner again for months after treatment has been administered. If some of them die meanwhile, the cause of death is apt to remain undetermined. Symptoms of greater or less severity due to intoxication by various anthelmintics are not uncommon.

In a recent paper, Paul D. Lamson, Harold W. Brown and Charlotte B. Ward discuss anthelmintics generally, dealing more fully with four of them, namely, tetra-chlor-ethylene, ficin, hexylresorcin (hexylresorcinol), and 6-n-hexyl-meta-cresol.[1] Lamson and his fellow investigators have not been able to find evidence of toxicity of tetra-chlor-ethylene to laboratory animals. This substance appears to be absorbed from the alimentary tract of man, and is apt to cause symptoms similar to those due to the absorption of alcohol or chloroform. It is probably about as effective against hookworm as carbon tetrachloride, and is ineffective against ascaris. As yet it is not known whether tetra-chlor-ethylene, like carbon tetrachloride, ever causes ascaris to migrate.

Ficin is a proteolytic enzyme obtained from the sap of *Ficus laurifolia*. It has a powerful action against ascaris and trichuris. It is non-irritating and non-toxic, providing there is no lesion of the intestinal mucous membrane. If such a lesion exists, ficin may cause erosion. This substance cannot be recommended for general use in the present state of knowledge concerning it.

Hexyl-resorcin was introduced as an anthelmintic by Lamson about two years ago. Of all drugs used as anthelmintics, hexyl-resorcin is the only one known to these investigators which is given with the object of being absorbed from the alimentary tract in the treatment of conditions other than helminthic infestation. This cannot be allowed to pass without comment. Oil of turpentine is an anthelmintic of sorts, and it is still prescribed in the treatment of pulmonary disease. Doubtless there are other examples among the hundreds of drugs used at various times as anthelmintics. Hexyl-resorcin in solution in olive oil has been used for some years as a urinary antiseptic, under the trade name of "Caprokol". It has been administered several times a day, in doses larger than the anthelmintic dose, for many months, to people in a very poor state of health, without causing any apparent intoxication. Approximately 30% of hexyl-resorcin taken by mouth is rapidly excreted in the urine as the ethereal sulphate; the remainder is excreted unchanged in the stools. Apparently none is retained in the organs. The excretion is so rapid that a high concentration in the body never occurs.

Hexyl-resorcin is of great value in the treatment of infestation with ascaris or hookworm. It is moderately effective against trichuris. In a small series of cases it was found to be lethal to enterobius. Several tapeworms have been removed by its administration; but Lamson and his fellow investigators are not prepared to make a statement regarding its value in the treatment of infestation with these parasites.

Hexyl-resorcin has two disadvantages. In the first place, it combines with protein, and is therefore relatively ineffective if taken when the stomach or intestines contain food. In the second place, it has a local irritating action. It causes slight irritation to the gastric mucosa, and, if taken in the ordinary crystalline form, has a corrosive action on the mucous lining of the mouth. It is inactive as an anthelmintic when given in oil; its poor solubility prevents its administration in water; and it decomposes gelatine capsules. It must therefore be prescribed as a sugar-coated pill. There is always a danger that children will suck or chew these pills.

Lamson, Brown and Ward have investigated several substances akin to phenol and resorcin, and have found that 6-n-hexyl-meta-cresol, a liquid, has

[1] *The Journal of the American Medical Association*, July 23, 1932.

practically no local irritating action and is apparently harmless to laboratory animals. They are not yet prepared to recommend its administration to man. They express the hope that it will be a "useful clinical substance and that further investigation of this group of phenols will bring forth an anthelmintic which is so safe, effective and cheap that it can be generally distributed throughout the world . . . so that finally, after sufficient education . . . people can rid themselves of these parasites by self-medication without danger of intoxication".

Apparently hexyl-resorcin is a very powerful anthelmintic, and, if carefully administered, a perfectly safe one. It should be of great value in private medical practice when adequate supervision can be exercised; but it is not suitable for "mass" treatment. According to Lamson, Brown and Ward, the oral lesions are superficial and quite harmless; but their appearance must be rather frightening. These investigators are to be congratulated on their efforts to discover the ideal anthelmintic; their success would be of almost incalculable value to the world. Pleasing features of their paper are their cautiously expressed conclusions and an absence of extravagant claims for the value of their discovery.

TRAUMATIC CHYLOTHORAX.

The effusion of chylous or chyloid fluid into the pleural sac is by no means a common event. Nevertheless, it is both of interest and importance, for some of the problems involved are obscure and the prognosis is serious. The subject has been historically reviewed by D. S. Macnab and E. P. Scarlett in an article describing a well studied example.[1] These authors have collected from the literature thirty-three case records of chylothorax following injury, and add another from their own practice. The majority of the patients had suffered injury other than wounds; most of the recorded injuries were due to crushing or a severe fall on the thorax, one to fracture of the clavicle, and several to wounds of the base of the neck.

A perusal of the case histories reveals some curious features. One of the most striking is the sudden onset of critical symptoms after a latent period of several days. The patient attended by Macnab and Scarlett fell eleven feet and struck his back on a piece of timber. He did not present any signs of serious illness, no fracture was discovered, and he was able to sit up and to go to the bathroom. Suddenly, six days after the accident, he became shocked and dyspnœic, with a subnormal temperature, cyanosis and sweating. The right side of the chest was found to be dull as high as the clavicle, and aspiration was performed, a large quantity of the characteristic milky fluid being withdrawn. At this stage was noted another of the remarkable features of this condition, the rapidity with which the fluid accumulates. In this instance 1,750 cubic centimetres were withdrawn early in the morning, no less than 2,000 cubic centimetres in the same afternoon, and during the following forty-eight days 35 litres of fluid were aspirated from this patient's chest. Progressive weakness was a prominent feature of the illness, which ended fatally two months after the causal accident. The authors draw attention to the distinction to be drawn between a true chylous exudate, due to leakage from the thoracic duct, and a chyloid fluid which contains emulsified fat from degenerated cells, whose opacity is due not to chyle, but to a lecithin-globulin complex. Their analyses of the pleural fluid in this case leave no doubt as to presence of chyle. After repeated aspiration it was found very difficult to obtain fluid, owing to the presence of large masses of coagulum, and thoracotomy was therefore performed. Another argument for this was the creation of a positive intrapleural pressure, but the provision of drainage in no way improved the patient's condition.

As regards the curious features mentioned, one cannot but be struck by the dramatic onset of symptoms in many cases. This has been ascribed to anaphylactic shock or to protein intoxication by some writers. The extraordinary profuseness of the exudate is not only interesting, but important, for, with a patient showing signs of frequently recurring distress, something must be done to give relief. Macnab and Scarlett do not mention gas replacement as a means of treatment. It is quite possible that the introduction of gas or air would prevent the sudden fluctuations in pressure attendant upon the evacuation of large pleural exudates and thereby tend to limit the leakage of chyle until such time as healing could occur. That free communication with a ruptured thoracic duct occurs was proved in the present instance by the demonstration of an actual tear *post mortem*.

Another difficult problem is that of the patient's metabolism. The asthenia and wasting are out of proportion to what might be expected. These patients often seem to go downhill more rapidly than the loss of lacteal fat would explain, and such measures as a diet rich in carbohydrate and fluids, and even the restoration of the chylous fluid to the circulation, appear to avail little. It would seem to be probable that something more serious than the mere escape of chyle is involved, for the mortality consequent on operative injury to the thoracic duct is but 10%, while in traumatic chylothorax it is nearly 50%. The authors ask whether the steady deterioration in the patient's condition is due to loss of fat, to the toxic action of chyle in the tissues, to interference with the whole lymphatic circulation, or to some breakdown in lipoid metabolism. Apparently no definite answer can be given. Here, then, we have a condition certainly rare, but of grave importance. Whenever it is recognized, it should be the subject of detailed study and should be reported in the literature, in order that knowledge may grow and treatment improve.

[1] *The Canadian Medical Association Journal*, July, 1932.

Abstracts from Current Medical Literature.

OPHTHALMOLOGY.

Sympathectomy and Retinitis Pigmentosa.

G. CAMPBELL (*The Canadian Medical Association Journal*, June, 1932) reports the results of excision of the superior cervical ganglion in a case of *retinitis pigmentosa*. The patient was a woman of twenty-four years, who gave a history of ocular trouble for eight years. Her vision became blurred after reading a short time. Her fields were restricted and she saw badly at night. Her colour vision was worse than formerly. The fundi were characteristic. Vision in each eye was $^{6}/_{12}$. After operation on the left side, the left pupil was contracted to three millimetres, an exophthalmos of four millimetres was observed, also a pseudo-ptosis. Four months later a change in the white field only was noticed on the left side, with a small sector in the temporal area of the right side. Later, there was further improvement in the white and green fields. Macular vision remained the same. Eleven months after the first operation, the right ganglion was removed, but no further improvement was noted. The author and the operator (R. E. McKechnie) are sufficiently optimistic that they intend to continue performing this operation as suitable cases present themselves.

The Ophthalmologist and Lighting Problems.

W. B. LANCASTER (*Archives of Ophthalmology*, July, 1932) believes that ophthalmologists should have some knowledge of defective illumination, not only when the illumination is obviously bad, but when it appears to be good but is in reality defective. The points to be considered are the quantity or brightness, secondly, the quality, colour or wave length, and thirdly, the distribution. A light of ten foot-candles gives the maximum visual acuity, if the surface looked at has good reflecting power. This produces a brightness of ten millilamberts. Some surfaces, such as dark cloth, reflect as little as 10% of light, in which case the illumination needs to be 100 foot-candles. When a system of lighting is found unsatisfactory, it is usually the distribution or the quantity that is at fault, rather than the quality or wave length. The eye dislikes light from a bright source, with intense shadows and an extreme range between the light and dark part of the room. To avoid points of high intrinsic brilliancy indirect lighting is employed. The light sources are hidden and the light is reflected into the room from the higher part of the wall and ceiling. If this is not feasible, the lights should be 30° above the line of sight of the worker. With indirect lighting it is important to make the quantity sufficient. It should be at least 17 foot-candles on the working table. The present standard is from one to three watts per square foot. For reading it is not good practice to have the light shining brightly on the book while the surrounding room is dim. Glare is excessive brightness of the whole or any part of the field such as to cause reduced vision or discomfort. Glare can be avoided by concealing the sources of light or sufficiently shading them.

Iridodialysis.

B. W. KEY (*Archives of Ophthalmology*, May, 1932) operated upon a boy of ten years for a large iridodialysis of one year's standing, associated with a partially absorbed traumatic cataract. The capsule and lens were divided with a Ziegler's knife and an attempt was made to fix the periphery of the iris into the iritic angle. As this failed, a keratome incision was made, the margin of the iris was drawn out and transfixed with a fine silk suture. The double armed suture was then carried through the sclero-conjunctival flap at the limbus and tied. Two sutures were then placed in the conjunctival flap for further support. The result was good. A similar operation was performed on another boy with iridodialysis in the lower nasal quadrant. The result in this case was a pear-shaped pupil. In commenting upon this operation the author states that it is neither difficult nor uncertain in its performance and that the hazard of operation is in direct proportion to the extent and location of the defect and should be measured in terms of the deformity and the probable improvement in visual efficiency.

Proptosis Due to Traumatic Aneurysm.

G. H. HANNA (*The Canadian Medical Association Journal*, June, 1932) reports a case of pulsating exophthalmos in a man of twenty years. He was injured by a pitch-fork which entered his left orbital cavity. On examination a few hours afterwards, the pupil was semi-dilated and inactive. Eye movements and fundus were normal; vision was confined to the counting of fingers. Two weeks later the following condition was noted: ptosis of upper lid, marked protrusion of the eyeball, complete ophthalmoplegia. Ten weeks after the injury pulsation was first discernible and a bruit was heard; the bruit ceased on pressure over the common carotid. The patient also complained of a fullness in the left ear, and by placing one end of a rubber tube in the patient's ear and the other end in his own, the author could hear the bruit. A diagnosis of carotid aneurysm was made and ligature of the internal carotid was performed. The proptosis decreased 30%, but the patient did not regain consciousness; the right side of the body was completely paralysed. The patient died forty-eight hours after ligature. Autopsy revealed a softened area in the left temporal lobe. The cavernous sinus was filled with blood clot, but no arterio-venous communication was detected.

Blindness from Methyl Alcohol and Lumbar Puncture.

G. H. MATHEWSON AND B. ALEXANDER (*The Canadian Medical Association Journal*, June, 1932) report the case of a man of thirty-four years, who drank two ounces of "rubbing alcohol" which contained 75% of methyl alcohol. The following morning he took a second drink of two ounces, and by noon his vision failed, so that soon he could see only shadows. Three days later there were slight dilatation of the pupils, hippus, sluggish light reflex and blurring of disks; vision was perception of hand movements only. The retinal vessels were normal. The systolic blood pressure was 118 and the diastolic pressure 78 millimetres of mercury. The next day 18 cubic centimetres of cerebro-spinal fluid were withdrawn by lumbar puncture and this was repeated on the three following days till 74 cubic centimetres had been removed. Vision began to improve after the second lumbar puncture. Thirty-five days after the first dose of alcohol the vision of both eyes was $^{6}/_{6}$. In the meantime the disks showed optic atrophy and narrowing of the retinal arteries. Bougers recommends gastric lavage, continued for four or five days. The author has seen many cases of methyl alcohol blindness, but never before had he seen a patient recover good vision. The actual change in the retinal cells is probably œdema; hence the value of lumbar puncture and pilocarpine sweats.

Myasthenia Gravis.

S. V. ABRAHAM (*Archives of Ophthalmology*, May, 1932) defines *myasthenia gravis* as a symptom complex characterized by sudden, intermittent and progressive weakness of a muscle or muscle group, often terminating in death and leaving little pathological changes. The age of onset varies from early childhood and senility, but is most common between twenty and fifty years. It is more common in females. Ptosis or diplopia is the first symptom in 40% of cases. Ptosis can be brought on or increased by exercise for a short time, a characteristic of the disease. Quincke's œdema, urticaria and rapid flushing reveal an unstable vasomotor mechanism. In making duction tests of ocular muscles with Maddox rod and prisms, it is found that, despite previous effort, the muscles immediately relax almost entirely on suspension of fusion. This condition does not exist in the myasthenic patient, as proved by several cases. The indications are that the muscles contract, but fail to relax promptly or completely, remaining in a prolonged contraction state. In these circumstances *myasthenia gravis* is not a true myasthenia.

OTO-RHINO-LARYNGOLOGY.

Streptococcus Hæmolyticus Carriers and Puerperal Sepsis.

W. R. Logan (*The Journal of Laryngology and Otology*, March, 1932), recording his observations on the effect of tonsillectomy on carriers of the *Streptococcus hæmolyticus*, refers to the observations of many recent workers that in a large proportion of the more serious cases of puerperal sepsis and in most of those occurring in the epidemic form, the *Streptococcus hæmolyticus* is the bacterial factor. J. Smith is quoted as having demonstrated that the serological type of streptococcus differed in various cases, but in a large proportion of cases that the type was the same in the throat of the medical attendant as in the uterus of the patient. A small series, members of the nursing staff of the Royal Infirmary, were tested by the author. Swabs were taken from the throats of nineteen nurses who had had their tonsils completely dissected out, and from nineteen control nurses who had not been operated on. *Streptococcus hæmolyticus* was isolated from seven, or 36·8%, of the normal control nurses, and five, or 26·3%, of the nurses whose tonsils had been removed by dissection. The author quotes Fraser as stating that "enucleation of the faucial tonsils does not get rid of the lingual tonsil at the back of the tongue nor of the small aggregations of lymphoid tissue on the lateral and posterior walls of the pharynx. This, of course, is quite apart from the question of the teeth. One cannot remove all the lymphoid tissue in the upper respiratory tract." The author concludes that the results of tonsillectomy do not justify its adoption if carried out for the sole purpose of ridding the throat of *Streptococcus hæmolyticus*.

Atrophic Rhinitis and Ozæna.

Simon L. Ruskin (*Archives of Otolaryngology*, February, 1932), in a paper dealing with the differential diagnosis and therapy of atrophic rhinitis and ozæna, states that these are two distinct diseases that have been considered throughout the literature as one and the same disease. The pathology of the two conditions is described and the author is of the opinion that ozæna is pathologically an obliterating endarteritis of the nasal mucosa and that atrophic rhinitis is the result of a chronic inflammatory disease characterized by round-cell infiltration, probably secondary to severe rhinitis. Operation for narrowing the nasal fossæ by extensive and destructive surgical operation has no rational basis anatomically, physiologically, pathologically or clinically, and is not based on a conception of the ætiology. The therapy for ozæna is essentially to be directed toward the blood vessels, and in considering an obliterating endarteritis occurring in youth, one must recognize that it represents a disturbance in equilibrium between vasoconstriction and vasodilatation. Ozæna can be improved by blocking of the vasoconstrictor fibres at the nasal ganglion. Atrophic rhinitis must be treated along non-specific lines, such as are used in chronic infections. The paper is illustrated by photomicrographs.

Chronic Middle-Ear Suppuration.

S. H. Mygind (*The Journal of Laryngology and Otology*, May, 1932), in a paper dealing with the indications for radical operation in chronic middle-ear suppuration, reviews 800 cases of chronic purulent *otitis media* in which 550 patients had been operated upon. He discusses the results of the complete radical operation compared with Bárány's partial radical operation and the plasticless radical operation. He draws attention to the frequency of relapses which follow this operation, and discusses the rôle played by cholesteatoma. The study of these cases confirms the author's opinion that the best results of treatment of chronic purulent *otitis media* do not follow the complete radical operation. The plasticless total radical operation should be abandoned. Bárány's partial radical operation cannot be entirely dispensed with. The indication is especially in cases with an apparently well preserved middle ear and good hearing, or in which hearing in the other ear is particularly bad. In the opinion of the author, the greatest difficulty lies in the treatment of children, and he insists that they be submitted to a comprehensive general medical examination, so that their family predispositions to ear trouble, the state of their homes and diet, may be investigated. Every effort should be made to invigorate their general well-being with the view to having the operation performed under the best possible conditions of resistance.

Pins at the Periphery of the Lung.

Chevalier Jackson and Chevalier Lawrence Jackson (*Archives of Otolaryngology*, June, 1932), in a paper entitled "Pins in the Periphery of the Lung", based on observations on forty-two cases, describe the treatment of foreign bodies which have invaded relatively minute bronchi. Fluoroscopic pathfinding and its indications are dealt with. The costo-phrenic bronchoscope and standard rotation forceps are used. The pin, when grasped by the shaft, can be buckled into the tube mouth, causing both pointed and head extremities to retreat so that the potentially dangerous advancing point is converted into a trailing point. The authors conclude that pins at the periphery of the lung are practically always lodged head downward. This may be due to the catching of the point and the tumbling over of the head end, and partly to the fact that the head end is heavier. The primary cause of pins in the lung is carelessness in putting pins in the mouth. The cause of the pin reaching the periphery is the pawl and ratchet-like action of the pin; the head is free to move downward during inspiratory elongation of the bronchi, and the point catches and resists upward movement during the bronchial shortening of expiration and cough. The limit of downward travel is the smallest bronchus that the head of the pin can enter. After the initial choking, gagging and coughing, there is, when pins are lodged at the periphery of the lung, a symptomless interval of a number of months. Sooner or later, however, suppurative changes, with productive cough and progressively increasing impairment of health, supervene, and a fatal ending may eventually occur if the pin is not removed. All pins at the periphery of the middle and lower lobes and descending branches of the upper lobes can be removed through the mouth by peroral costo-phrenic bronchoscopy. The ascending branches of the upper lobes present great difficulties, but fortunately their invasion by pins is exceedingly rare.

Osteomyelitis of the Skull and Nasal Accessory Sinus Disease.

Abraham O. Wilensky (*Archives of Otolaryngology*, June, 1932) discusses cases of osteomyelitis of the skull which are found to complicate acute and chronic disease of the nasal accessory sinuses. Only infections caused by the ordinary pyogenic bacteria are considered. The infection that leads to the fatal result is usually post-operative and occurs within two or three days after an operation for disease of the nasal accessory sinuses. The diploe of the frontal bone is invaded by the organisms and an osteomyelitis is set up. The infection spreads thence; on the one hand, it passes through the outer table, giving rise to subperiosteal abscesses beneath the scalp; on the other hand, it passes inward and may subsequently give rise to extradural or subdural abscess, general meningitis, cerebral abscess or a thrombosis of the longitudinal or other sinuses. The *dura mater* is affected in practically all cases, and the pachymeningitis may remain localized for a long time. Thrombosis occurs chiefly in the lateral or longitudinal sinuses. The invasion of a large venous sinus is often manifested by emboli with distant metastases. Pneumonia or bronchopneumonia frequently occurs. When the osteomyelitis becomes evident, it is necessary to operate, and the operation should be as extensive as necessary. The cranial bones should be resected beyond the limits of the lesion. If the wound continues to granulate and if the temperature remains high, sequestra are present, and they must be completely removed. There is a large mortality. The patient succumbs to one or more of these conditions with or without manifestations of a general blood infection, as evidenced by the presence or absence of a positive blood culture, after a varying number of weeks or months, ranging to one or more years.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the B.M.A. Building, Adelaide Street, Brisbane, on August 5, 1932, DR. E. S. MEYERS, the President, in the chair.

The Median Pressure and Hypertension Moyenne Solitaire Syndrome.

DR. CYRIL SHELLSHEAR and DR. CLIVE H. SIPPE read a paper entitled: "The Median Pressure and Hypertension Moyenne Solitaire Syndrome: Some New Aspects of Oscillometry" (see page 412).

DR. N. W. MARKWELL thanked Dr. Shellshear and Dr. Sippe for their paper. It showed a great deal of careful research through the literature and they had also investigated a series of cases themselves. The subject was an important one. Dr. Markwell had used Pachon's oscillometers only for the last fourteen years. The diastolic pressure had originally been placed lower down at the bottom of the curve, as Dr. Shellshear had pointed out. Later on, Pachon came to the conclusion that the diastolic pressure was just below the longest oscillation, namely, the point which was called the median pressure in the paper. Dr. Markwell had followed this lead. Therefore, all the records of the diastolic pressure thus recorded by Pachon's oscillometer were really records of the median pressure, but about five to ten millimetres below it. It was therefore more accurate when the oscillometer was used to get away from the term diastolic pressure and to record the exact median pressure, as suggested in the paper. Dr. Markwell had had three oscillometers in fourteen years and the instrument had never let him down. This could not be said of other string instruments on the market. He agreed with Dr. Shellshear that the mercury instruments should be replaced by Pachon's oscillometer or other oscillometer which could be equally relied upon. Fourteen years previously there had been only one bag in the cuff which was placed over the wrist, and the results obtained then were not comparable with the results obtained by the mercury manometer. However, the double cuff used now allowed this instrument to be compared in its results with the Riva-Rocci instrument. Dr. Markwell agreed that when the median pressure was close to the systolic pressure, it was of great importance. He quoted a case of a male patient with a large heart, who suffered from cardiac asthma at night, but who was quite well in the day time; he had seen the patient in consultation. The medical practitioner in charge of the case reported that the systolic blood pressure was not high, being only 160 millimetres of mercury, and he did not report anything unusual about the diastolic blood pressure. The Pachon instrument showed clearly that the systolic pressure was 160 and the median pressure 150 millimetres. The electrocardiograph confirmed the serious condition of the patient, so-called arborization block being present, and the patient died soon afterwards. In the case of a raised median pressure in a young person, one should be careful in one's diagnosis. Dr. Markwell stated that such a patient was not necessarily "a heart case" already; he might be a "potential heart case". He thanked Dr. Shellshear and Dr. Sippe for their extremely instructive paper.

DR. ELLIS MURPHY congratulated Dr. Sippe and Dr. Shellshear on their paper. He was struck with the great amount of work involved in it. One point was the actual mechanical difficulty of taking the charts; each chart represented up to ten or twenty readings and it would take an enormous amount of time. Also the finding of the actual pressures was not altogether a simple matter. Dr. Murphy had found in his notes that the readings did vary and quoted cases to this effect. He had not found the mean pressure such a criterion as many authors claimed. He found that sometimes a patient's clinical condition had improved with a mean pressure that had gone up. Patients with arteriosclerosis and those with marked Graves's disease gave a high oscillometric reading.

DR. E. S. MEYERS said that the subject matter of the paper was very important, as disease of the heart and arteries was among the most frequent causes of death. A number of preconceived ideas were upset. How was the general practitioner going to get on? Would he have to discard his present instrument? Dr. Meyers wished to ask if any work had been done as to deciding whether patients were good "operative risks" or not. This should help a lot with patients in the fourth and fifth decades, who were not in good physical condition.

Dr. Sippe, in reply to Dr. Markwell, said that twelve years earlier the term "diastolic" had been used by Pachon, but he had now termed this the median dynamic pressure. With regard to alterations in the size of the heart in young persons and in a potential heart case, the important point was not so much the size of the heart, but the amount of function present. The result of the exercise tolerance test was important in these cases. If there was a high response of the mean pressure to exercise, the outlook was worse. It was not certain yet whether electrocardiographic changes were always evidence of a bad prognosis.

In reply to Dr. Murphy, Dr. Sippe said that when the oscillations were high, the taking of readings was very difficult with the oscillometer, but the new instruments had no valve to operate and were much easier to read. One could put the figures down without making a graph. Importance was attached to the results of the exercise tolerance test, even if the mean pressure were near the systolic. If the exercise tolerance test gave a good result, the prognosis could be considered good, even if the mean pressure were five to ten millimetres below the systolic. If the result of the tolerance test was poor, the height of the mean pressure did not matter so much. Quite a number of patients with hypertension gave high oscillations, but the smallness of oscillations did not necessarily give any definite prognosis or significance. In Graves's disease one would expect high oscillations. This appeared to be a possible diagnostic point, but in a study of a number of cases they were disappointed, as the height of the oscillometric index gave no indication as to the presence of hyperthyreoidism.

Dr. Shellshear, in reply, said that there was no need to discard the mercury manometer. There was an instrument that was extremely reliable, being made by Mr. Aitken, of Annerley Road, South Brisbane, without a manometer, but with an attachment for one, mercury or otherwise.

Dr. Sippe would show them the new Vaquez instrument, named a kymometer, on which a spring manometer was a fixture.

So far, no testing of patients before surgical operation had been done. The work was very new and the last article was only out in the Australian mail of a fortnight before. Dr. Shellshear had started the work simply because it was of interest to him and seemed of great importance. He hoped to do work on the peripheral vessels which would be of interest in conditions such as vagotonia, sympatheticotonia, *endarteritis obliterans et cetera*. Dr. Sippe had replied to most of the points brought up.

Medical Societies.

THE MELBOURNE PÆDIATRIC SOCIETY.

A MEETING OF THE MELBOURNE PÆDIATRIC SOCIETY was held at the Children's Hospital, Carlton, on June 8, 1932, DR. R. WEBSTER, the President, in the chair. The meeting took the form of a series of clinical demonstrations.

Purpura Hæmorrhagica.

DR. LIONEL HOOD showed a girl of twelve years, who gave a history that she had been quite well except for occasional epistaxis during the past few years, becoming more frequent lately. Following a severe epistaxis in April, 1932, she was admitted to the ward and the nostril was plugged. A week later the hæmorrhage recurred and

a bleeding point on the septum was cauterized, but this arrested the bleeding only for a few hours. Examination at this time revealed a pale exsanguinated girl with, apart from the nose, no evidence of hæmorrhage from any of the mucous membranes. On the abdomen and trunk were a few recent petechial spots, the spleen was just palpable, and there was a faint apical systolic murmur, probably hæmic in origin. Blood examination showed that there was a severe secondary anæmia, coagulation time was normal, but bleeding time was prolonged to twenty minutes and the platelets were practically absent.

The patient was given a transfusion of thirteen ounces of citrated paternal blood and the hæmorrhage ceased. One week later she had another severe epistaxis and became desperately ill. At this stage no platelets were found on examination of her blood. She was again transfused with fifteen ounces of citrated blood, the hæmorrhage ceased, and for ten days she progressed quite well. Then her gums began to bleed, more petechial spots appeared, but the epistaxis did not recur. The bleeding became very severe and was uncontrollable by local treatment, so a transfusion of sixteen ounces of citrated blood from a friend was given. Complete hæmostasis followed and she was well for fourteen days, when she had some further oozing from the gums, with more petechial spots, but the bleeding was slight and responded to local treatment. Blood examination revealed that the bleeding time had now returned to normal, but platelets were still very scanty.

A culture from the tonsils revealed hæmolytic streptococci. Dr. Hood said that the patient had had no further hæmorrhages and the spleen was impalpable. The patient was presented for an opinion as to whether splenectomy was advisable.

DR. F. KINGSLEY NORRIS said that this would seem to be a case of secondary thrombocytopenic purpura rather than the true primary Werlhof's disease, in which the typical findings in the blood of *purpura hæmorrhagica* persisted during the remissions. It was in this latter type that splenectomy was indicated. In Dr. Hood's case the bleeding time was now normal, and perhaps the platelets would recover too. Probably the hæmolytic streptococcus from the tonsil was the cause, and, if so, splenectomy should not be undertaken.

DR. J. W. GRIEVE said that in many cases of apparently true primary thrombocytopenic purpura, once the hæmorrhage had ceased, there did not seem to be any tendency for the bleeding to recur, and, as in all probability that would hold for this case also, he did not think splenectomy should be undertaken.

Erythrœdema.

DR. LAWRENCE STOKES presented a female child, aged ten months, with typical symptoms of erythrœdema. The mother had been able to breast feed her child for some few weeks only and the infant then was given boiled cow's milk. The symptoms of the disease had been present for five weeks before the child was presented. The mother also stated that the child had had no orange juice for the previous three months.

As a point of interest the wrists and knees were examined by X rays and the plates showed typical early bony changes characteristic of scurvy. There was no change in the appearance of the gums, nor was there any tenderness over the tibiæ.

Dr. Stokes stated that he showed the patient for two reasons. Firstly, on account of the interesting combination of erythrœdema and scurvy, and secondly, to stimulate discussion directed towards the further investigation of these cases of "pink disease". In connexion with the scurvy condition, he stated that during the previous three weeks he and Dr. Boyd Graham had seen three cases of undoubted scurvy in the same clinic at the Children's Hospital. He naturally blamed the economic depression, and suggested that probably during the coming winter many more cases would be seen.

With regard to further investigation of cases of erythrœdema, both Dr. Reginald Webster and Dr. Stokes stated that Dr. Penfold, the Director of the Baker Research Institute of the Alfred Hospital, and Miss Butler, of the same institute, were anxious to obtain tonsil material from early cases. In view of the possibility that this condition might be of infective origin, they hoped to carry out some bacteriological investigation to prove or disprove this hypothesis, using the new Kendall culture medium.

Branchial Fistula.

DR. D. O. BROWN showed a girl, aged five years, with a branchial fistula. The very small external opening had been noticed soon after birth and had been discharging intermittently for the last two months. The discharge appeared to have been associated with recent colds and tonsillitis. Examination revealed a healthy type of child, but with enlarged and septic tonsils. The external opening of the fistula was demonstrated about 2·5 centimetres (one inch) above the clavicle, at the anterior border of the left sternocleidomastoid muscle. The fistulous track could be felt as a fibrous cord passing upwards from the external opening deep into the neck. The act of swallowing caused upward retraction of the opening. Radiographs taken after lipiodol injection showed the track to have some bulbous enlargement about the level of the angle of the mandible and then to pass on towards the pharynx in attenuated form, ending near to, or in, the pharyngeal wall. Attempts to inject methylene blue through the fistula into the pharynx had failed. Tonsillectomy had been performed two weeks earlier, and the discharge had ceased for ten days, but had then recurred. The patient was shown, first, on account of the relative rarity of the condition in children of this age and to invite opinions as to the advisability of surgical intervention. Complete excision of the track in a small child presented very definite intrinsic difficulties. It was to be remembered that such fistulæ arose from remnants of usually the second branchial cleft, thus necessarily passing between the internal and external carotid arteries to reach the pharyngeal wall in the region of the supratonsillar fossa. Embryologically, they should never actually communicate with the pharynx, as the cleft membrane in the human embryo was not perforated at any stage in development. Many authorities claimed that the communication commonly described was artificial.

DR. W. KENT HUGHES said he had operated on a number of occasions on children for a similar condition. He had usually injected the fistula with methylene blue to define its limits. He had invariably reached the end of the track well before the pharyngeal wall, and thought that most of such fistulæ ended in the region where the lipiodol in this case showed a bulbous enlargement of the track. He advised operation, as the discharge would probably persist.

DR. H. D. STEPHENS had had very variable results from his operations for branchial fistulæ. Some had healed well, but others had required repeated operations in after years. Nevertheless he thought it advisable to excise such fistulæ. He had not found methylene blue satisfactory because of its diffusing into the surrounding tissues so easily, and thought that a more viscous coloured fluid would be useful. He had commonly found at operation that these fistulæ became so attenuated as to be virtually lost before the pharynx was reached, and had never dissected one right to the pharyngeal wall.

DR. J. G. WHITAKER mentioned that hæmorrhage was an occasional operative difficulty, and pointed out that with a child it would be quite safe to tie the common carotid artery if hæmorrhage were otherwise uncontrollable.

Thymic Tumour.

DR. MOSTYN L. POWELL showed a baby, aged five months, who was brought to hospital when three months old because of vomiting, often projectile, after every feeding since birth. He was a full time baby, delivered with instruments, and was entirely breast fed, every four hours. Despite the vomiting, he was gaining weight at the rate of six ounces per week until two weeks prior to coming to hospital, when he gained only two ounces per week. The family history was irrelevant. On examination

no abnormality could be detected in the nervous system, no pyloric tumour could be felt, nor was there any enlargement of the spleen or lymph glands. The case was regarded as one of pylorospasm and the patient was treated with *liquor atropini sulphatis*, 0·3 mil (five minims) before each feeding, and for two days there was no vomiting at all, but it soon recurred and the diagnosis was reconsidered. There had been no cough, cyanosis, dyspnœa nor any signs of mediastinal pressure, except that the baby showed a tendency, when held in the mother's arms, to lie in an opisthotonic position.

A radiological screen examination was made to exclude diaphragmatic hernia. None was seen, but there was a definite supracardiac loss of translucency extending into the upper zone of the right side of the chest. A lateral film showed that this mass was in the anterior mediastinum. It was regarded as an enlarged thymus, although unusual in being decidedly asymmetrical. A barium emulsion passed uninterruptedly into the stomach, so that there was no relationship between this mass and the vomiting.

The child was given deep X ray therapy at the Melbourne Hospital, consisting of three exposures of twenty-nine minutes each at intervals of a week. Each dosage was of one hundred and fifty international Röntgen units, at a distance of 37·5 centimetres (fifteen inches), through 0·5 millimetre of copper and 4·0 millimetres of aluminium.

Weekly X ray photographs since that time showed the characteristic response of this simple lymphoid hyperplasia to deep X ray therapy. There was a rapid return to an apparently normal supracardiac shadow within three weeks. The baby had since greatly improved, there was no hyperextension of the neck, and the vomiting had ceased, though he still required the atropine before his feedings. As the vomiting responded to atropine and as there was no obstruction to a barium emulsion, there was probably no relationship between the enlarged thymus and the vomiting. The patient was presented to exemplify Holt's statement that these simple thymic hyperplasias were symptomless, were relatively frequent, and would spontaneously retrogress during infancy. This case was detected only during investigation of a coincident pylorospasm. It also bore out Holt's belief that these cases were quite unrelated to *status lymphaticus* (if such a condition existed), for the baby bore no resemblance to the pallid, fat, flabby type associated with this condition, nor was there any enlargement of the other lymphoid structures of the body.

Rickets.

DR. J. W. GRIEVE showed a male infant, aged one year and five months, admitted with a history of cough and fever for three days. The family had come to Australia from Greece four years ago. There were ten brothers and sisters, all alive and well. One child had died at the age of two days. There was no history of rickets in the other children, though a history was difficult to obtain, as the mother could not speak English. This child had been breast fed for five months, then boiled milk and water in equal parts with added sugar were given. No orange juice was added till the child was nine months old, and then only one teaspoonful was given *per diem*. No cod liver oil was given at all. The only additions to the milk and water had been broth, cereals and bananas.

The father was a fruiterer and, so far as one could gather, the child was kept indoors most of the time. He had attended the out-patient department irregularly since the age of two months for umbilical hernia and retarded development.

Examination on admission to the ward revealed skeletal changes typical of rickets. The skull was natiform, with frontal bossing, the fontanelle was widely open, there was craniotabes in the region of the lambdoid suture, and only four teeth had erupted on each side, the first one appearing after the age of one year. There was a rickety rosary, with splaying of the lower ribs and enlargement of all the epiphyses, with the characteristic X ray findings. The abdomen was protuberant and the liver and spleen were readily palpable. He had profuse head sweats and general hypotonia. No tetany was manifest. He was being treated by having his diet elaborated with eggs, bacon fat, fish and liver, by being given cod liver oil, one teaspoonful three times a day, and by general ultra-violet light exposures.

He was presented because such a severe grade of rickets was very rare in Melbourne, particularly in Australian children. It was noteworthy that this child was a Greek.

DR. COLIN MACDONALD said that in a recent *Archives of Disease in Childhood*, Dr. Kempson Maddox had reported that in a large series of unselected children in Sydney he had detected evidence of rickets in more than 50%. According to their clinical and radiological standards, the incidence in Melbourne children would not even approach that figure. Either rickets must be much more common in Sydney or, what was more probable, there must be a different standard for clinical and radiological diagnosis of the disease.

Correspondence.

"AVERTIN."

SIR: My attention has been directed to Dr. Idris Morgan's letter in your issue of August 27, 1932.

With your permission may I deal *seriatim* with certain elementary points of vital importance to patient, anæsthetist and surgeon, which I am afraid your correspondent has failed to appreciate.

1. The induction and maintenance of surgical anæsthesia demands the absorption of a given amount of ether. Factors influencing absorption are concentration of the vapour and pulmonary ventilation.

2. An excessive concentration of ether vapour tends to lead to the development of laryngeal spasm and the occurrence of pulmonary complications. On the other hand, undue dilution delays induction and prolongs the stage of excitement.

3. By artificially increasing the respiratory minute-volume (with the aid of carbon dioxide or a Lidwill or similar machine) an increased amount of ether can be absorbed in a given time at a lower concentration. Conversely, depression of the respiratory centre leads to inadequate pulmonary ventilation and the consequent administration of increasing concentrations of ether vapour.

It is remarkable that most writers on "Avertin" have fallen into the error of utilizing respiratory rate to assess pulmonary ventilation, whereas the more important factor is respiratory amplitude.

4. "The average patient, if properly handled, can be kept fully surgically anæsthetised with from three ounces to four ounces of ether per hour after he has been properly induced" (R. W. Hornabrook).

Although enthusiasts claim that "Avertin" enables the consumption of ether to be cut down to minute quantities, no Australian figures have been published to prove this contention.

In the cases reported in your issue of July 23, 1932, the amounts of ether used were: (i) seven ounces; (ii) four ounces; (iii) six ounces *plus* chloroform, one fluid drachm; (iv) six ounces, for operations not exceeding sixty-five minutes. Incessant coughing characterized induction in cases (i) and (iii).

In the detailed analysis of twenty-one cases of "Avertin Rectal Anæsthesia," published by Dr. Ellen M. Balaam in THE MEDICAL JOURNAL OF AUSTRALIA of June 20, 1931, twelve of the cases received approximately seven and a half ounces of "C_1E_5" mixture, an average of a little over five-and a half fluid drachms per case.

5. "Most observers stress the depressant action on respiration and in my own cases the respiration has always been shallower than normal, but the rate usually increased to about twenty-four per minute . . . The complication most to be feared is respiratory failure. This is unlikely to occur if 'Avertin' is used as a basal anæsthetic only, that is, when the dose does not exceed 0·1 gramme per kilogram" (C. C. Coghlan).

In the editorial of May 21, 1932, one reads: "In one large hospital several deaths have occurred after the administration of 'Avertin'." Such fatalities were by no means restricted to one hospital and certain of them appear to be frank cases of fatal narcotic poisoning. It is to be regretted (or "deprecated") that these cases have not been reported.

In Dr. Balaam's series, cyanosis was noted in five of the twenty-one cases reported. It is common knowledge that cyanosis does not commence to be apparent until the oxygen saturation of the arterial blood has fallen to 80% or 85%.

The rôle of anoxæmia in damaging the central nervous system and myocardium is another story.

6. Muller Overholt and Pendergrass have stated that the normal pulmonary vital capacity is reduced 60% during operations on the upper part of the abdomen and 40% during operations on the lower abdomen. These same observers have shown that a minor degree of apneumatosis, which they term pulmonary hypoventilation, can be demonstrated by radiography as a normal occurrence after upper abdominal operations.

In view of the above data one is interested to know by what logical method Dr. Idris Morgan arrived at his conclusion that "Avertin" was not a causal factor in the production of the pulmonary complications referred to in your issue of July 2, 1932, and the grounds for his statement, "it now transpires that this conclusion was quite unwarranted".

As your correspondent prefaces his letter by the remark "sweeping statements unsupported by facts and details . . . succeed in clouding the issue only", unless he can bring forward evidence such as respiratory exchange, blood gas estimation or some such tangible data in connexion with "Avertin" basal narcosis, he has condemned his own contribution as "unworthy to be classed as scientific". A truly interesting position for one who casts reflections upon the observation powers of others.

With regard to the part played by "Avertin" in the production of pulmonary complications, subsequent to surgical manœuvres in the upper part of the abdomen, the final court of appeal must be an investigation into pulmonary ventilation during the course of "Avertin"-open ether anæsthesia.

Meanwhile, the opinion "that open ether anæsthesia after preliminary narcotization with 'Avertin' is contraindicated for operations on the upper part of the abdomen in men" stands.

Yours, etc.,

H. Rutherford Darling.

229, Macquarie Street,
Sydney,
August 29, 1932.

Sir: Is it not time for the disasters that are stated by you to be occurring with "Avertin" anæsthesia to be reported in full, *minus* names and addresses, in the journal? It seems hardly fair or scientific to state that "Avertin" anæsthesia can be dangerous and leave it at that; the point that should be emphasized is how it can be dangerous, with illustrations from the disasters aforementioned. I think if detailed reports were obtained of these cases, it would be found that the administration had not been exactly according to the directions issued by the manufacturers, or that contraindications had not been carefully considered. From my own experience of about sixty cases, I think it is a very satisfactory anæsthetic. I have always used it as a basal anæsthetic without morphia, and have not hesitated to use sufficient ether to supplement it (the amount of ether used is seldom more than two ounces for an appendicectomy).

Dr. C. B. Howse, my partner, considers it to be a remarkable advance in anæsthesia and is enthusiastic about it. There is no doubt as to its popularity with patients, and if ether is used before the skin incision, the surgeon can have no complaint as to its efficacy. The anæsthetist, after a little experience, can decide for which patients the full dose is necessary (0·1 cubic centimetre to a kilogram). There are many cases in which a smaller dose is equally effectual. The preparation and administration certainly take a little time, but that is a small price to pay for the patient's relief from anxiety and delightfully comfortable return to consciousness.

Yours, etc.,

J. T. Paton.

Orange,
New South Wales,
August 30, 1932.

[The editor of a journal cannot compel anyone to report a case, be the outcome successful or fatal. An appeal has been made in the editorial columns of this journal for reports of fatalities following the use of "Avertin". Anonymity would deprive a report of much of its value. With Dr. Paton we are anxious to discover why disasters have occurred. If individual practitioners are unwilling to attach their names to the report of a fatality, there is no reason why a report should not be made by the staff of a hospital. Such a report would carry weight.—Editor.]

Sir: The marked antagonism and protagonism that has characterized the correspondence about "Avertin" in recent numbers of the journal is difficult to explain. What is there about "Avertin" that differentiates it from opium and its derivatives, chloryl hydrate, "Sodium amytal", "Luminal" and a host of other drugs given for the same purpose and with similar effect. The literature here and abroad is filled with particulars of the results of premedication by "Luminal", "Sodium amytal", "Nembutal" and paraldehyde, to mention a few only, and there has resulted no shower of heated correspondence. Perhaps some of the confusion is caused by some protagonists using or misusing the word "anæsthesia" in their letters, when a careful perusal shows that it has been used merely for premedication. The term "basal anæsthetic" is very little more correct. Many of us have used premedication with morphia, hyoscine *et cetera* prior to both general and local anæsthesia for years, and some of us have recently used paraldehyde, "Luminal", "Sodium amytal" and other drugs for the purpose of preventing the psychic shock of anæsthesia and operation, but we have not referred to "morphia anæsthesia", "'Sodium amytal' anæsthesia" *et cetera*.

There is a demand, and a justifiable one, by the public for something that will insulate them against the mental stress that precedes surgical intervention, and it is our duty to supply this if we are of the opinion that this can be done without addition to the risks and with advantage to the patient. Our first attempts took the form of morphia. This proved to have certain risks and disadvantages. Then paraldehyde was introduced with considerable success. Recently "Luminal", "Sodium amytal", "Avertin" and many similar drugs have been extensively used. My own experience has been practically confined to "Sodium amytal", and I have found it an advantage in suitable cases. My aim has been to exhibit the minimum dose that would give analgesia during the time before operation and the commencement of anæsthesia proper, and I think that it is because the correct and intelligent appreciation of the field for their use has been lost sight of by some enthusiasts that we have had fatalities. Fatalities have been reported with "Avertin", but these have been described as due to "'Avertin' anæsthesia", which may mean that attempts have been made to operate during coma induced by the drug alone. If attempts are made to render the patient sufficiently comatose to operate by any of the other drugs without inhalation anæsthesia, there would certainly be fatal cases, but I have seen no reports of deaths caused by overdosage with the other drugs.

There is no doubt that "Avertin", like the other drugs, can be safely used for the purpose indicated, that is, to abolish memory of the time preceding operation, and that in such doses it is probably safe. The aim should be to arrive at the minimum dose that will give the above

results, and any attempt to achieve a depth of coma sufficient to allow operations is fraught with dangers in the case of "Avertin", just as with opium or any other irrecallable drug used for this purpose.

Yours, etc.,

H. C. E. DONOVAN.

195, Macquarie Street,
Sydney,
September 19, 1932.

THE EFFECTS OF SOLAR RADIATION.

SIR: It was with very great interest that I read Dr. Molesworth's article, *vide* THE MEDICAL JOURNAL OF AUSTRALIA, August 13, 1932, in which he urged the adoption of a wide-brimmed sombrero type of hat for general use in Australia.

Might I make so bold as to champion the same cause; whereas Dr. Molesworth is concerned with the ætiology of cancerous growths from exposure to solar radiation, I am concerned with the effects upon and through the eyes. I am more and more convinced that we descendants of the Nordic race are as yet unfitted to tolerate the undiluted solar rays and the effects they produce upon the retinæ, even though it be accepted that most of the ultra-violet rays are absorbed by the cornea, aqueous and lens. Many individuals are unaffected, it must be admitted, but there is a large section of the community which suffers by the exposure. The more common incidental ailments are: glare asthenopia, frontal and occipital headache, irritability of temper, asthenia, slipshod concentration. In addition, there is the unsightly wrinkling of the brow and frowning aspect. Since women began to wear berets and the brimless hat of the present day vogue, it is quite common to hear patients complaining of some of the above symptoms, and if the advice be given and followed, namely, to have some protection in the way of a hat brim, relief is invariably attained.

I could enlarge upon the thesis, but scarcely feel there is occasion to. Common sense, comfort and experience urge me to join Dr. Molesworth in seeking to evolve a rational sized hat brim and its use in this sunlit and glaring atmosphere. I would go so far as to say that the brim need not necessarily be sombrero wide, but should have a slight downward incline to compensate for width; it must not fold upwards, or the shading value of breadth is sacrificed.

Yours, etc.,

GRANVILLE WADDY.

227, Macquarie Street,
Sydney,
August 15, 1932.

THE PSYCHOLOGY OF A PRIMITIVE PEOPLE.

SIR: In an otherwise fair review of my recent book "The Psychology of a Primitive People", appearing in your issue of April 9, were several points of criticism to which I should like to reply.

The reviewer seems to object to claims that my book was in any sense a pioneer psychological study and remarks: "A great deal of work has been done on the psychology of the Australian aboriginal." No doubt he refers to the invaluable studies of Spencer, Gillen, Howitt and Roth, which truly contain an immense amount of psychological material, but, at the same time, could hardly be described as psychological studies. In the same way the observations of great explorers, such as Captain Cook or Sir George Grey, contain much anthropological material, but they are not usually numbered among the anthropologists.

As a matter of fact, the pioneer Australian anthropologists did not approach their problem from the specifically psychological point of view. For example, they were but indirectly concerned with the motivation underlying the aborigines' social customs, nor did they consider those customs in the light of special adaptability to the conditions of their physical environment. My concern, on the other hand, was directly with the adaptive values of their social laws and expedients for these were to my mind proofs of considerable intelligence if viewed from that angle. I did not rely entirely on the mental test results to determine aboriginal mental status, yet this approach also was necessary. Considering only the native Australian's social expedients, he ranks high in intelligence. Considering his individual adaptability to the kind of problems which civilized life presents, he ranks very low. Both methods of appraisal are necessary.

As is well known, the work of the early observers of aboriginal life was descriptive rather than interpretative. The reviewer may, of course, disagree with my interpretation of the various customs or may dissent entirely from the relationship that I have tried to work out between the peculiar demands of the aborigines' environment and the societal bonds which they have fashioned for themselves. He may be scornful of my attempts to explain why the native Australian behaves as he does, but to suggest that these psychological interpretations are to be found in the work of previous observers is to betray either bias or ignorance of what those anthropologists wrote. Occasionally, it is true, they did draw some psychological conclusions, as, for example, when Spencer states that the nakedness of the aborigines was due to their inability to conceive of the use of skins as clothing, or when Howitt goes to the other extreme and credits the natives with the realization of the ill-effects of incestuous matings and assigns that as the psychological basis for the exogamic system. With both these conclusions the social psychologist is forced to disagree. Surely one may admire to the utmost the work done by these pioneer anthropologists, but it is too early yet to proceed to their scientific canonization or to the assumption that they have preempted the whole field.

I am glad, however, that the reviewer does not dispute my claim to priority as regards the psychological examinations—and this was really as far as my claim extended. He may put these examinations at little value; but to my mind the demonstration was at least useful in showing that the aborigines of the centre had superior tested intelligence to those of the north-west coastal regions, from which fact might be drawn conclusions of considerable interest to psychology. Considering that the reviewer missed this point and also the fact that he is quick to discover that I do not know the difference between the doctrine of the virgin birth of Christ and the Immaculate Conception, I suspect that he is a much better theologian than a psychologist. He is probably correct when he says that immediately I enter the realms of theology I lose myself. Here at least I shall make no claims for priority, for I have yet to meet the theologian who is not also thoroughly and completely lost in his subject. Though undoubtedly a wretched theologian, I may not be the worse scientist thereby.

I share the regret of the reviewer that the book is not more scientifically satisfactory. I, too, am disappointed in the number of my subjects, the extent of my observations and the adequacy of my methods, but the answer to such criticisms is that we need more of this kind of work, not less, and I sincerely hope that a too critical or ungracious reception of their efforts will not discourage Australian psychologists from doing their part in the anthropological field.

There is much to be done, and the opportunity is passing swiftly. Indefatigible as the labours of the Australian anthropologists have been, and excellent though the results of recent and current anthropological research undoubtedly are, the student of primitive psychology will find the available data regarding the aborigines all too scanty. I would therefore repeat, despite the reviewer's criticism, that this is "a virgin field in science, offering the promise of exceptional returns for labour expended in it". I had hoped that my study might have served as an encouragement to Australian psychologists to get on with the work of study-

ing the aborigines from every possible angle rather than to consider the field already exploited—the job satisfactorily done.

Yours, etc.,

S. D. Porteus,

Professor of Clinical Psychology.

The University of Hawaii,
Honolulu, Hawaii,
August 18, 1932.

POLIOMYELITIS.

Sir: As the person responsible for the introduction of the use of serum in Australia in March, 1925, may I say that at the outset we were very careful not to use it in doubtful cases, as we wished to establish its use on a firm basis and the supply was very scanty. Now that supplies are more plentiful, I think it is wise to use it in doubtful cases, just as we use antitoxin in doubtful diphtheria. I agree with Dr. Helms that the cell count is most important, but I don't agree with her statement that "we cannot restore the anterior cornual cells that are destroyed", for it implies that in every case some such cells are destroyed. If that were true, our results would be less encouraging. I think that explosive destruction of cells by hæmorrhage only occurs in the more virulent cases and in the mild type that is allowed to walk about. In order to eliminate quickly the strangling effects of exudate, might I suggest the use of diathermy to the cervical and lumbar enlargements as soon as temperature has fallen. I can certainly testify to its use as suggested by Bordier, of Lyons. Why I deplore the fussy investigation of muscles involved and the useless and expensive treatment of prolonged so-called muscle reeducation is because both of these must increase the congestive condition. To lay such stress upon muscle movements as against complete rest as possible is to resurrect the absurd theory of Dr. Colin Mackenzie, that poliomyelitis is an affection of the muscles and that involvement of the nervous system has nothing to do with the case.

In view of the fact that so many cases of poliomyelitis exhibit increasing weakness many years after the onset, it is instructive to record the following case.

A girl of twenty-two, who had been operated upon at six years of age for severe *talipes equino-cavus* sixteen years ago, came to me a few months ago with a history of increasing weakness in her left leg (the one operated upon) and a definite *equino-cavus* in her right foot. Three months ago there was an inch difference in thigh measurement three inches above the upper margin of the patella. Today the left thigh has picked up that inch and her left calf is half an inch greater in measurement. She doesn't get so tired when walking and can play basket ball. Although I am not going to be dogmatic and put the improvement down to diathermy, it is suggestive that, though she failed to improve after eighteen months of exercises, in the last three months she has made such improvement.

Yours, etc.,

W. Kent Hughes.

22, Collins Street,
Melbourne,
September 21, 1932.

Obituary.

LESLIE JAMES LAMROCK.

Dr. Leslie James Lamrock, whose death was announced in a recent issue of this journal, was born at Grose Vale, New South Wales, in October, 1868. His father was the late William Lamrock, grazier. He went to school at Cooerwull Acadamy, Bowenfels, and matriculated at an early age. As a boy he was fond of cricket and football and won several cups for athletic sports. He maintained his love of sport throughout his life. After spending one year at the University of Sydney, he went to Edinburgh. He studied assiduously and gained a medal for anatomy. In 1891 he graduated as Bachelor of Medicine and Master of Surgery and obtained the degree of Doctor of Medicine in 1893. His thesis for this degree was on rickets. After holding a resident appointment in Yorkshire, he returned to Australia and took up practice at Waverley, Sydney. He practised there till his death.

Lamrock was known among his colleagues as a careful practitioner who acted from the highest motives. His patients respected and loved him. He had many interests apart from medicine. He owned several racehorses and was a regular attendant at the racecourse. He was a keen golfer and was an honorary life member of the Waverley Bowling Club. He was a Vice-President of the Royal Agricultural Society, Sydney. He was for many years a member of the Waverley Municipal Council, and more than once occupied the mayoral chair. It was while he was Mayor that the Waverley Town Hall was built, and he was responsible for many of the charming flower and grass reserves in the locality. He was fond of swimming and surfing, and was elected by the Waverley Council as life member of the Bondi Swimming and Surf Clubs. He purchased a property in the country and devoted his attention to apple growing and sheep breeding. He was fond of dogs and had a fine breed of fox terriers. Four of his dogs followed his funeral. He was a man who will be missed by a large circle of personal friends and a larger circle of patients.

University Intelligence.

THE UNIVERSITY OF MELBOURNE.

The Registrar of the University of Melbourne reports that at the last meeting of the Council of the University, the following matters respecting the Medical School were dealt with.

Professor Wood Jones having been granted leave of absence to take charge of the Union Medical School at Pekin for six months, Dr. W. E. A. Hughes Jones was appointed Acting-Professor of Anatomy.

Dr. Hilda Rennie, Lecturer in the Department of Bacteriology, was granted leave of absence until the end of the year, and her appointment was then terminated on health grounds.

Notice was received that the late Dr. A. V. M. Anderson, who at the time of his death was Lecturer in Medical Ethics, had left to the University two-ninths of his residuary estate. What this will amount to is not yet known.

A bequest of £1,200 by the late Mrs. Ada Haddon, for cancer research, was received.

THE UNIVERSITY OF ADELAIDE.

During the past twelve months the following changes have been instituted by the Council of the University of Adelaide.

1. The following institutions have been proclaimed schools of medical instruction for students of the Adelaide University: (*a*) The Mareeba Babies' Hospital, (*b*) The Northfield Consumptive and Cancer Home, (*c*) The Bedford Park Sanatorium.

2. As part of the course of instruction in medical diseases of children, arrangements have been made for demonstrations to be given at The Mareeba Babies' Hospital and at a welfare centre.

3. Establishment of Tutorship in Infectious Diseases: A tutor is to give lectures and clinical demonstrations at the Metropolitan Infectious Diseases Hospital at Northfield.

4. A commencement has been made with the erection of a new building for the Department of Chemistry. The building is estimated to cost £26,000.

5. The University Council accepted an offer received from the Section of Clinical Medicine of the British Medical Association (South Australian Branch) to donate each year a prize of £5, to be awarded to the medical student who shall obtain the greatest merit in the medical group section of the final examination.

Books Received.

HYPNOTISM, by Alexander Cannon, M.D.; 1932. London: William Heinemann (Medical Books) Limited. Crown 8vo., pp. 60. Price: 2s. 6d. net.

THE BRITISH PHARMACOPŒIA, 1932, PUBLISHED UNDER THE DIRECTION OF THE GENERAL COUNCIL OF MEDICAL EDUCATION AND REGISTRATION OF THE UNITED KINGDOM. London: Constable and Company, Limited. Medium 8vo., pp. 763.

Diary for the Month.

OCT. 4.—New South Wales Branch, B.M.A.: Council.
OCT. 5.—Victorian Branch, B.M.A.: Branch.
OCT. 5.—Western Australian Branch, B.M.A.: Council.
OCT. 6.—South Australian Branch, B.M.A.: Council.
OCT. 7.—Queensland Branch, B.M.A.: Branch.
OCT. 7.—New South Wales Branch, B.M.A.: Annual Meeting of Delegates of Local Associations.
OCT. 10.—New South Wales Branch, B.M.A.: Organization and Science Committee.
OCT. 11.—New South Wales Branch, B.M.A.: Ethics Committee.
OCT. 13.—New South Wales Branch, B.M.A.: Clinical Meeting.
OCT. 14.—Queensland Branch, B.M.A.: Council Meeting.
OCT. 18.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
OCT. 19.—Western Australian Branch, B.M.A.: Branch.
OCT. 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.
OCT. 26.—Victorian Branch, B.M.A.: Council.
OCT. 27.—South Australian Branch, B.M.A.: Branch.
OCT. 27.—New South Wales Branch, B.M.A.: Branch.
OCT. 28.—Queensland Branch, B.M.A.: Council.

Medical Appointments.

Dr. K. B. Shallard (B.M.A.) has been appointed Government Medical Officer at Boonah, Queensland.

* * *

Dr. G. Atkinson (B.M.A.) has been appointed Public Vaccinator for Macarthur, Victoria.

* * *

Dr. R. M. Downes (B.M.A) and Dr. W. W. S. Johnston (B.M.A.) have been appointed members of the Masseurs' Registration Board of Victoria.

* * *

Dr. E. J. T. Thompson (B.M.A.) has been appointed Acting Inspector-General of the Insane, Acting Medical Superintendent of Claremont, Greenplace and Whitby Falls Hospitals for Insane, Lemnos Soldiers' Mental Hospital, and Heathcote Mental Reception Home; also Acting Inspector-General of the Institution for Inebriates, Western Australia.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi.

ECHUCA DISTRICT HOSPITAL, ECHUCA, VICTORIA: Resident Medical Officer.

HOBART PUBLIC HOSPITAL, HOBART, TASMANIA: Junior Resident Medical Officer.

LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officer.

ROYAL NORTH SHORE HOSPITAL OF SYDNEY, NEW SOUTH WALES: Honorary Dermatologist.

THE BRISBANE AND SOUTH COAST HOSPITALS BOARD, QUEENSLAND: Honorary Officers.

THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Junior Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor", THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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